IMAGING CHARACTERISTICS IN PATIENTS WITH NORMAL PRESSURE HYDROCEPHALUS AT TERTIARY HEALTH CARE HOSPITAL

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DECLARATION

In the following pages is presented a consolidated report of the study **"Imaging characteristics in patients with Normal Pressure Hydrocephalus at Tertiary Health Care Hospital"** a cross sectional study, on cases coming to Radiodiagnosis outpatient Department at Sree Mookambika Institute of Medical Sciences, Kulasekharam from 2016-2017. This thesis is submitted to the Dr. M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulations for the award of MD Degree examination in Radiodiagnosis.

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ABBREVIATIONS

NPH	-	Normal Pressure Hydrocephalus
EI	-	Evans Index
CA	-	Callosal Angle
SF DILATATION	-	Dilation of the Sylvian fissure
DESH	-	Disproportionately enlarged subarachnoid space
DWMH	-	Deep white matter hyperintensities
PVH	-	Periventricular hyperintensities-
WM	-	White Matter
VP SHUNT	-	Ventriculoperitoneal Shunt
SAH	-	Subarachnoid Hemorrhage
CT	-	Computed tomography
MRI	-	Magnetic resonance imaging
CSF	-	Cerebrospimal fluid
MRE	-	Magnetic resonance elastography
CVD	-	Cerebrovascular disease
PwMR	-	Perfusion weighted magnetic resonance
CST	-	Corticospinal tract

ABSTRACT

ABSTRACT

INTRODUCTION

Normal pressurehydrocephalus (NPH) is a syndrome found in the elderly, which is characterized by the clinical triad of gait disturbance, dementia, and urinary incontinence without overt signs and symptoms of elevated intracranial pressure. NPH has been estimated to account for upto10% of cases of dementia and is significant because it is treatable by ventriculoperitoneal shunting.

NPH can be idiopathic or can be secondary. The secondary causes are: traumatic brain injury, meningitis, subarachnoid hemorrhage (SAH) or intracranial surgery. Patients with Idiopathic NPH respond better to treatment than secondary NPH.

AIMS AND OBJECTIVES

- To describe the various imaging patterns helpful in the diagnosis of normal pressure hydrocephalus

MATERIALS AND METHODS

It is a prospective cross-sectional study. Inpatients and outpatients of age group more than 40 years of age, of both gender (males and females) diagnosed with normal pressure hydrocephalus as per consensus criteria were referred to Department of Radiodiagnosis from the department of Neurology of Sree Mookambika Institute Of Medical Sciences were enrolled in the study. The patients underwent Magnetic resonance imaging / computed tomography study of brain. The study period was 18 months.

RESULTS

40 patients were included in this study according to consensus criteria. Computed tomography (CT) and Magnetic resonance imaging (MRI) show ventricular enlargement disproportionate to cerebral atrophy, with associated ballooning of frontal horns, periventricular hyperintensities, thinning and elevation of the corpus callosum, and widening of temporal horns without evidence of hippocampal atrophy in NPH.

CONCLUSION

Although diagnosis can be made based on CT findings alone, MRI is more accurate for disclosing associated pathologies (such as cerebrovascular disease) and for detecting NPH typical signs of prognostic value, besides avoiding exposure to ionizing radiation. MRI is the best modality to image anatomical changes and can further support the diagnosis with CSF flow studies.

KEY WORDS: normal pressure hydrocephalus, neuroimaging, magnetic resonance imaging, computed tomography, cerebrospinal fluid pressure, shunt surgery

INTRODUCTION

INTRODUCTION

Normal pressure hydrocephalus is characterised by a triad of dementia, gait ataxia and urinary incontinence without overt signs and symptoms of elevated intracranial pressure. The triad is famously referred to as Hakim's triad for NPH.[1-4]

NPH can be idiopathic or can be secondary. The secondary causes are: traumatic brain injury, meningitis, subarachnoid hemorrhage (SAH) or intracranial surgery. The exact pathology of idiopathic normal pressure hydrocephalus (iNPH) is still unclear.[4]

The underlying pathophysiological mechanism suggests impaired cerebrospinal fluid (CSF) flow within the ventricles or subarachnoid space or both, defective CSF absorption through the arachnoid granulation and impaired intracranial vascular compliance.[4]

There is no accepted pathological criteria for postmortem diagnosis of iNPH. While potentially causative abnormalities like arachnoid fibrosis, have been reported in brain autopsies of some patients who were diagnosed with iNPH but it has not been studied in a systemic manner and therefore it cannot verify a clinical diagnosis of iNPH.

According to international guidelines, the following are CT or MRI criteria which are decisive for NPH diagnosis: ventricular enlargement disproportionate to cerebral atrophy (Evans index > 0.3), with associated ballooning of frontal horns; periventricular hyperintensities; corpus callosum thinning and elevation. Callosal angle between 40º and 90º and widening of temporal horns not fully explained by

hippocampal atrophy; and aqueductal or fourth ventricular flow void; enlarged Sylvian fissures and basal cistern, and narrowing of sulci and subarachnoid spaces over the high convexity and midline surface of the brain.[5]

The newer MRI applications provides indications of abnormal CSF flow by using Proton Density MRI, Phase Contrast MRI, Radionuclide cisternography documenting CSF flow void, ventricular reflux and hyperdynamic flow. Various Advanced MRI measurements can be performed as adjuncts to conventional clinical sequences in patients suspected of having NPH: Volumetric MRI, Diffusion Tensor Imaging, Arterial Spin Label and Phase Contrast Imaging.[6]

iNPH is an important cause of motor disturbances and cognitive impairment in elderly patients , the social and economic burden attributable to iNPH cannot be ignored. Therefore, any kind of research effort aimed at improving the understanding of the epidemiology of iNPH is important.[7]

NPH is the only form of dementia that can be treated with ventricular shunting. According to some studies, the only evidence pointing to a diagnosis of NPH is good response to ventricular shunting. Normal pressure hydrocephalus is increasingly recognized as a treatable cause.[8]

AIM OF THE STUDY

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- To describe the various imaging patterns helpful in the diagnosis of normal pressure hydrocephalus

SCIENTIFIC JUSTIFICATION

SCIENTIFIC JUSTIFICATION

The prevalence of Normal Pressure Hydrocephalus remains uncertain, with reported rates varying from 1.3 per million to 4 per 1000, depending on diagnostic criteria for Normal Pressure Hydrocephalus and populations sampled. Recent estimates quote an annual incidence of 1.8 per 100,000. The similarities in the symptoms and the abnormal size of the ventricles between Normal Pressure Hydrocephalus and other disorders like Alzheimer's and Parkinson's diseases makes Normal Pressure Hydrocephalus patients difficult to identify, leading to misdiagnosis. This is a real challenge for the medical community. The distinction is clinically important because Normal Pressure Hydrocephalus is one of the few treatable causes of dementia.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

` Normal pressure hydrocephalus is a syndrome of dementia, gait ataxia and urinary incontinence without any apparent signs and symptoms of elevated intracranial pressure. The triad is famously referred to as Hakim's triad for NPH.[1-4]

Nityanand, Miskin et al conducted a study in 2017 titled "Diagnosis of Normal-Pressure Hydrocephalus: Use of Traditional Measures in the Era of Volumetric MR Imaging.[9] The purpose of the study was to assess the diagnostic performance of the callosal angle (CA) and Evans index (EI) and to determine their effectiveness in comparison to the automated volumetric methods in clinical radiology.[10,11] He performed Magnetic resonance (MR) examinations before surgery in 36 shunt-responsive patients with normal-pressure hydrocephalus (NPH) and MR examinations of age and sex-matched patients with Alzheimer disease (n =34) and healthy control volunteers (n = 36) were studied. The result of the study was that the model that used CA and EI demonstrated 89.6% – 93.4% accuracy and average area under the curve of 0.96 in differentiating patients with NPH from patients without NPH. The regression model that used volumetric predictors of gray matter and white matter was 94.3% accurate. He concluded that CA and EI may serve as a screening tool to help the radiologist differentiate patients with NPH from patients without NPH.

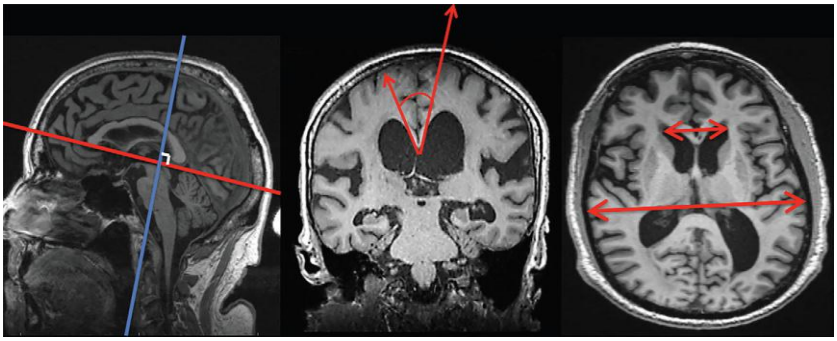

Fig. 1 MRI TIWI showing callosal angle and evans index measurement*

*Sample sagittal (left), posterior coronal (middle), and midtransaxial (right) segmentation masks overlaid on T1-weighted MR images of shunt-responsive NPH, definitive AD, and HC volunteers. These images show gray matter, white matter, ventricular, and hippocampal regions.[9]

Johan Virhammar et al conducted a study in 2014 titled "The callosal angle (CA) measured on MRI as a predictor of outcome in idiopathic normal-pressure hydrocephalus"[12]. This method was presented by Ishii et al.[13] The aim of this study was to compare the CA in shunt responders with that in nonresponders and clarify whether the CA can serve as a predictor of the outcome. Preoperative MRI brain scans were evaluated in 109 patients who had undergone shunt surgery for iNPH during 2006–2010. The patients were examined clinically before surgery and at 12 months postoperatively. Shunt responders had a significantly smaller mean preoperative CA compared with nonresponders: 59° (95% CI 56°–63°) versus 68° (95% CI 61°–75°) ($p < 0.05$). A CA cutoff value of 63° showed the best prognostic accuracy. He concluded that the preoperative CA is smaller in patients whose condition improves after shunt surgery and may be a useful tool in the selection of shunt candidates among patients with iNPH.

Masaaki Hashimoto et al conducted a study in 2010 titled Diagnosis of idiopathic normal pressure hydrocephalus is supported by MRI-based scheme: a prospective cohort study. Patients aged between 60 and 85 years with one or more of symptoms (gait, cognitive, and urinary problems) and MRI evidence of ventriculomegaly and tight high-convexity and medial subarachnoid spaces received VP shunt using the height/weight-based valve pressure setting scheme.[14] The full analysis set included 100 patients. A favorable outcome was achieved in 69.0% and

80.0% were shunt responders. When measured with the iNPH grading scale, the one-year improvement rate was 77.0%, and response to the surgery at any evaluation point was detected in 89.0%. He concluded that the MRI-based diagnostic scheme is highly useful. Tight high-convexity and medial subarachnoid spaces, and enlarged Sylvian fissures with ventriculomegaly, defined as disproportionately enlarged subarachnoid space hydrocephalus (DESH), are worthwhile for the diagnosis of iNPH.

William G. et al conducted a study in 1991 titled "Association of Deep White Matter Infarction (DWMI) with Chronic Communicating Hydrocephalus: Implications Regarding the Possible Origin of Normal-Pressure Hydrocephalus".[15] The purpose of this MR study was to determine if the two diseases demonstrated a statistical association.[16] Four groups of patients were evaluated: 20 patients shunted for presumed NPH (group 1); 35 consecutive patients with MR findings and clinical symptoms consistent with the diagnosis of NPH (group 2); 78 consecutive patients with patchy periventricular hyperintensity, that is, presumed DWMI (group 3); and 62 consecutive patients over age 60 without MR findings of communicating hydrocephalus (group 4), who served as a control for groups 1 and 2. The results were as follows, In group 1, 19 (95%) had at least mild DWMI and 12 (60%) had marked DWMI. In group 2,32 (91 %) had at least mild DWMI and 20 (57%) had marked DWMI. This compares with the control patients in group 4, 41 (66%) of whom had at least mild DWMI and 15 (24%) of whom had marked DWMI. There was a statistically significant association between DWMI and NPH ($p < .001$). In group 3, 63 (81 %) had at the least mild communicating hydrocephalus and 24 (31 %) had moderate communicating hydrocephalus.[17] A chi-square demonstrated a statistically significant association between the two diseases ($p < .05$).

Tomas Radovnicky et al conducted a study in 2015 titled "Disproportionately enlarged subarachnoid space hydrocephalus presence in patients with idiopathic normal pressure hydrocephalus".**[18]** He retrospectively analysed 1, 5 T MRI in 27 iNPH patients before surgery and MRI in 24 healthy controls. He assessed tight high convexity and medial subarachnoid space, dilatation of Sylvian fissure and focal dilatation of sulci. Patient outcome was measured by iNPH grading scale one year after surgery. In the group of 27 iNPH patients, we have found DESH presence in 15 cases (55, 6%), all 15 patients were shunt responders. In the group of 12 iNPH patients without DESH 4 of them did not respond to surgery (33, 3%). He concluded that DESH appearance on MRI is supportive for the iNPH diagnosis, but it should not be used as a single predictor.

ETIOLOGY

William G. Bradley et al in 2000 published a paper titled "Normal Pressure Hydrocephalus: New Concepts on Etiology and Diagnosis".[2] While many researchers have worked on the etiology of idiopathic NPH, no single theory is accepted widely. Ventricular enlargement can occur when the transmantle pressure, ie, the difference in pressure between the ventricles and the subarachnoid space, is increased. Decreased CSF resorption increases transmantle pressure. CSF resorption in NPH is definitely abnormal, as shown by the saline infusion test. Many researchers consider that CSF absorption occurs at the level of the arachnoidal villi (microscopic) or arachnoid granulations (macroscopic), while some other authorities feel that a substantial amount of CSF resorption occurs at the brain parenchymal level, ie, the transcapillary or transvenular level.[19] The fact that histologic analysis of the leptomeninges in idiopathic NPH fails to show fibrosis suggests upstream

obstruction and lends credence to the increased venous resistance theory.[20] Dr. Bradley mentions that the theory proposed by Bateman in this issue suggests that the initiating event in NPH is the increased transvenular resistance in the territory of the superior sagittal sinus. Since this could lead to ventricular enlargement and decreased blood supply in the same territory, it is an enticing theory-it encompasses the two major abnormalities in NPH.

William G. Bradley et al conducted a study in 1991 titled "Association of Deep White Matter Infarction (DWMI) with Chronic Communicating Hydrocephalus: Implications Regarding the Possible Origin of Normal-Pressure Hydrocephalus". A large body of experimental work has been performed to support the hypothesis that periventricular brain injury can lead to decreased periventricular tensile strength and subsequent ventricular dilatation.[21] This was previously thought to be due to intraventricular CSF pulsations, it may result from inward expansion of the brain during cardiac systole. In the normal brain, the intraventricular CSF pressure rise during systole reflects arterial inflow followed quickly by venting of blood from the cortical veins and venting of CSF from the convexity subarachnoid space and the lateral and third ventricles through the aqueduct.[22] During systole, the brain normally expands outward (compressing cortical veins and convexity subarachnoid space) and, to a lesser extent, inward (compressing the lateral ventricles). Inward motion of the lateral ventricles produces the CSF flow void in normal individuals. Should a significant percentage of the spin echoes be acquired during systole (i.e., systolic pseudogating), the flow void would be increased, even in normal individuals. Thus, an increased CSF flow void should not be used by itself as evidence of communicating hydrocephalus. In early communicating hydrocephalus, the ventricles eventually expand such that the convexity

subarachnoid space and the cortical veins are compressed. Since late systolic venting of venous blood is no longer possible, there is greater inward systolic motion of the lateral ventricles, the intraventricular CSF pulse pressure rises, and the CSF flow void is increased.[23] As postulated by Hakim et al., the resulting tangential periventricular shearing forces may then lead to further ventricular enlargement and the symptoms of NPH. Whether DWMI causes NPH or vice versa, arteriolosclerosis of the long penetrating arteries is likely to be involved. This may explain why both NPH and DWMI are diseases of elderly patients.[24] In younger patients with severe multiple sclerosis, periventricular demyelination may also lead to decreased tensile strength and progressive ventricular enlargement in the pattern of communicating hydrocephalus. Ventricular enlargement in patients with multiple sclerosis may not be purely central atrophy.

Michael Kiefer et al in 2012 studied on "The Differential Diagnosis and Treatment of Normal-Pressure Hydrocephalus"[8] explained the pathophysiology. There is a consensus that the imbalance of CSF production and resorption in NPH is not due to overproduction, and that the resistance to CSF outflow (Rout) is often elevated.[25] In the early phase of the disease, the intracranial reserve capacity appears to be low, as reflected by such measures as the craniospinal compliance or the pressure-volume index (PVI). It remains unclear, whether changes in compliance or Rout are contributory causes or simply epiphenomena of the condition. The frequent combination of NPH with cerebrovascular disease and Alzheimer's disease makes it attractive to consider models in which these three entities are causally related; for example, in one model, all three are caused by a loss of the Windkessel effect in the skull base arteries. This loss of elasticity may be either primary (e.g., due to atherosclerosis) or secondary, a consequence of low craniospinal compliance

impeding the expansion of the arteries at the skull base. The result is that higher compressive stress and greater shearing forces develop in the brain parenchyma. Tissue damage and loss ensue mainly in the periventricular areas because of the physical and physiological differences between superficial and deep (periventricular) brain tissue.[26] This focal brain damage manifests itself as ventriculomegaly without any need to model a static pressure gradient from the inner to the outer CSF spaces. A further consequence of loss of the Windkessel effect is a lowering of cerebral blood flow, which explains the common simultaneous occurrence of iNPH and cerebral hypoperfusion; the latter, in turn, lowers CSF turnover.[27] It has been hypothesized that low CSF turnover impairs the clearance of toxic metabolites and thereby contributes to the pathogenesis of Alzheimer's disease.

NEUROIMAGING TECHNIQUE

Jan A. L. Vanneste published a study in 1999 titled "Diagnosis and management of normal-pressure hydrocephalus". In classical NPH, CT shows moderate or severe ventricular enlargement out of proportion to cerebral atrophy and includes ballooned frontal horns and enlarged temporal horns without evidence of hippocampal atrophy.[28] Moderate cortical atrophy and diffuse ischemic white matter lesions are unfavorable predictors of postsurgical improvement but do not definitely exclude the diagnosis of shunt-responsive NPH.[29] Holodny et al. observed that marked widening of the sylvian fissures and cortical sulci may paradoxically decrease after a shunt in patients with shunt-responsive NPH. They hypothesized that this is due to CSF accumulation at the convexity and not to cerebral atrophy and concluded that patients should not be denied a shunting procedure solely on the

basis of focally dilated fissures and sulci.[30] The size of hydrocephalus can be quantified by assessing the frontal horn ratio, being the maximal frontal horn ventricular width divided by the transverse inner diameter of the skull at the same level. A minimum of 0.32 is required for considering NPH, but most NPH patients have ratios of 0.40 or higher.[31] Frontal and occipital periventricular lucencies (PVLs) in NPH have been attributed to transependymal CSF absorption. In fact, many shunt-responsive patients have either no PVLs or have PVLs due to spongiosis, decreased density of nerve cells, or ischemic lesions. Differentiating the causes of PVLs is possible by measuring the proton relaxation times on MRI.

Magnetic Resonance Imaging

MRI is the best neuroimaging technique for evaluating patients with presumed NPH. It contributes to the differential diagnosis by allowing the assessment of the mean cross-sectional volume of the hippocampal body and the degree of dilatation of the perihippocampal fissures. Atrophy of the hippocampus is a marker of Alzheimer's disease while in NPH the reduction in the hippocampal mass is due to temporal horn dilatation.[32] An advantage of MRI is that T2-weighted images may show a "CSF flow voiding sign (CSFVS)" consisting of a decreased MRI signal, mainly in the aqueduct, which is correlated with the velocity of pulsatile CSF flow. In shunt-responsive communicating NPH, the aqueductal CSF flow velocity may be increased, but the significance of this finding is still unclear as in recent studies the correlation between the degree of CSFVS and postsurgical outcome is low It is also remarkable that postsurgical improvement does not necessarily result in a decreased CSFVS. In some patients sagittal MRI may show a focal corpus callosum impingement which may be an additional argument for

considering impaired CSF circulation as the cause of ventricular enlargement. The high sensitivity of MRI for detecting periventricular and ischemic white matter lesions and the increased incidence of such lesions in NPH may pose a diagnostic dilemma because ischemic white matter lesions do not necessarily preclude improvement after a shunt. Hence the problem is to decide whether white matter abnormalities are coincidental ischemic lesions in true NPH or rather a MRI manifestation of subcortical arteriosclerotic encephalopathy or Binswanger's disease. There is now common agreement that when white matter ischemic lesions are pronounced, a shunt is very rarely effective.

Cisternography

Isotope cisternography initially seemed a promising technique for assessing the CSF circulation. In NPH a disturbed CSF flow consists of a so-called "reversed pattern" with stasis of the CSF in the ventricles for 48 h or longer. With the advent of CT, CT cisternography was also used to demonstrate disturbed CSF circulation with a similar reversed CSF flow.[33] Cisternography is now considered by most clinicians to be an unreliable predictive test, with a few exceptions. It has been shown that cisternography does not add to the diagnostic accuracy of combined clinical and CT criteria.

Measurements of cerebral blood flow and metabolism

Studies of cerebral blood flow using single photon emission CT, xenon-CT, transcranial Doppler sonography of the middle cerebral artery, and measurements of regional cerebral metabolism with positron emission tomography have shown decreased CBF and metabolism in NPH.[34] This is most obvious in the frontal and periventricular areas, but some investigators have also found decreased CBF in the

temporal and parietal cortical areas. Several investigators have assessed the reactivity of cerebral circulation in presumed NPH and have found that preserved reactivity predicts a good prognosis after shunt placement. This might be explained by the fact that in these patients periventricular ischemia is still reversible, and that decreasing the pressure on the ventricular walls would lead to an improved periventricular microcirculation.[35] There are several problems in evaluating the results of CBF studies. These include the differences in techniques used for assessing the CBF, inconsistent results on the topography of impaired CBF, a variable or even no correlation between changes in CBF and outcome after CSF removal or a shunt, and recurrence of decreased CBF without relapse of clinical signs. As the predictive value of CBF measurements is quite variable, this test is not generalizable but may be valuable for some clinicians in whose hands CBF measurements seem helpful in selecting the right patients for a shunt.

RECENT ADVANCES

William C. Bradley et al in 1996 conducted a study titled "Normal-Pressure Hydrocephalus: Evaluation with Cerebrospinal Fluid Flow Measurements at MR Imaging".[36] He studied 18 patients with NPH underwent routine MR imaging and CSF velocity MR imaging before ventriculoperitoneal (VP) shunting. The calculated CSF stroke volume and the aqueductal CSF flow void score were compared with the surgical results. All 12 patients with CSF stroke volumes greater than 42 ıuL responded favorably to CSF shunting. Of the 6 patients with stroke volumes of 42 uL or less, 3 improved with shunting while 3 did not. He concluded CSF velocity MR imaging is useful in the selection of patients with NPH to undergo shunt formation. Other studies by Enzmann et al have found that preliminary MR imaging

studies showing brain motion are useful to understand the radial shearing motion of the cortex caused by ventricular expansion which could lead to dementia.[37,38]

Kaspar-Josche Streitberger et al. in 2010 published a paper titled "In vivo viscoelastic properties of the brain in normal pressure hydrocephalus".[39] MR elastography (MRE) uniquely allows the measurement of viscoelastic constants of the living brain without intervention.[40,41] He studied on 20 patients with idiopathic and secondary NPH were examined by cerebral multifrequency MRE and compared with 25 healthy volunteers. Viscoelastic constants related to the stiffness (m) and micromechanical connectivity (a) of brain tissue were derived from the dynamics of storage and loss moduli within the experimentally achieved frequency range of 25–62.5 Hz. In patients with NPH, both storage and loss moduli decreased, corresponding to a softening of brain tissue of about 20% compared with healthy volunteers. This loss of rigidity was accompanied by a decreasing a parameter, indicating an alteration in the microstructural connectivity of brain tissue during NPH.[42] This disease-related decrease in viscoelastic constants was even more pronounced in the periventricular region of the brain. The results demonstrate distinct tissue degradation associated with NPH.

J. Forner Ginera et al conducted a study in 2012 titled Quantitative phase-contrast MRI study of cerebrospinal fluid flow: a method for identifying patients with normal-pressure hydrocephalus.[43] The aim was to evaluate the use of phase-contrast MR imaging to diagnose normal pressure hydrocephalus (NPH) and differentiate it from other neurological disorders. The study included 108 subjects, of whom 61 were healthy controls and 47, patients; in the patient group, 19 had cerebrovascular disease (CVD) and 28 had NPH. All patients underwent a phase-

contrast MRI study and several CSF flow and velocity parameters were measured at the aqueduct of Sylvius. Maximum diastolic velocity, mean flow, and stroke volume showed statistically significant differences that could be used to distinguish between NPH and CVD patients.[44] Stroke volume and mean flow showed no false positive results and successful classification rates of 86% and 79%, respectively. He concluded that phase-contrast MR imaging is a useful tool for the early diagnosis of patients with NPH.[45] CSF flow quantitative parameters with morphological features in a MR study, enable us to differentiate between NPH and CVD patients.

Hertel et al conducted a study in 2003 titled "Is a combination of Tc-SPECT or perfusion weighted magnetic resonance imaging with spinal tap test helpful in the diagnosis of normal pressure hydrocephalus". The aim was to evaluate the combination of spinal tap test (STT) with cerebral perfusion measurement assessed either by Tc-bicisate-SPECT (Tc-SPECT) or perfusion weighted MRI (pwMRI), or both, for a better preoperative selection of promising candidates for shunt operations in suspected idiopathic normal pressure hydrocephalus.[46] He examined 27 consecutive patients with a standard clinical protocol. The results of these examinations were compared preoperatively for each patient and correlated with postoperative clinical outcome after shunt surgery. The results of SPECT and pwMRI correlated in 92 % of the patients (11 of 12). It is concluded that a combination of clinical assessment with SPECT or pwMRI is helpful in the preoperative selection of patients for shunting procedures with suspected NPH syndrome.[47] This combination is a minimal invasive and objective test modality that is superior to STT alone.

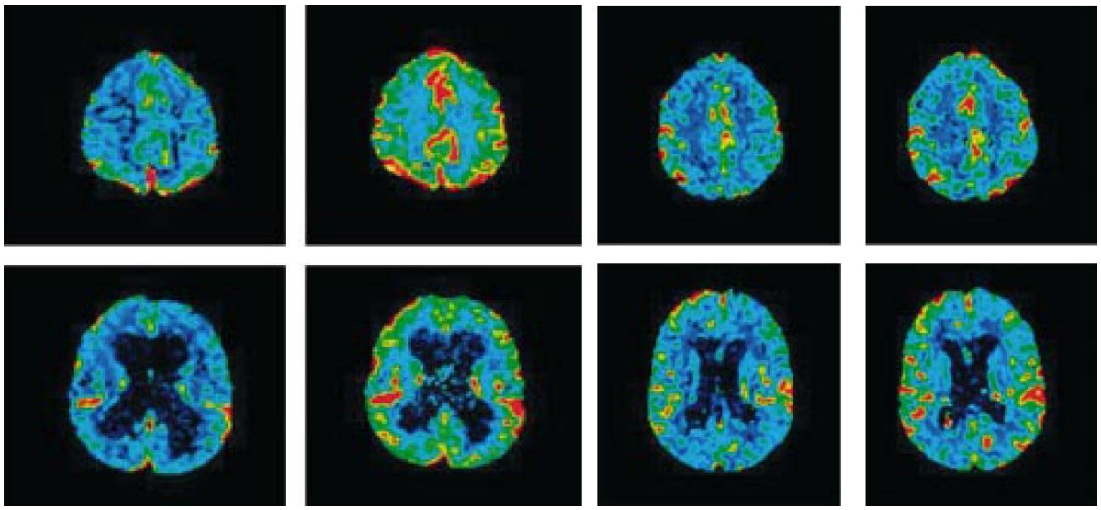

Fig. 2 Perfusion weighted MRI in a patient with increasing rCBV through spinal tap test. Before (left) and after (right) spinal tap test. Perfusion weighted MRI before (left) and after spinal tap test (right). There is no significant change in rCBV through spinal tap test. (Hertel et al 2003)

Nakanishi et al conducted a study in 2012 titled "Microstructural changes of the corticospinal tract in idiopathic normal pressure hydrocephalus: a comparison of diffusion tensor and diffusional kurtosis imaging".[48] The goals of this study were to examine the usefulness of diffusional kurtosis imaging (DKI) for assessing microstructural changes in the compressed corticospinal tract (CST) among patients with idiopathic normal pressure hydrocephalus (iNPH).[49] 11 patients with iNPH, who underwent 3-T magnetic resonance imaging, including DKI before surgery, were recruited. 6 age matched, healthy subjects served as the control group. Mean diffusional kurtosis (DK) and axial diffusion kurtosis were significantly lower in iNPH patients. However, apparent diffusion coefficient, fractional anisotropy, and axial eigenvalue were significantly higher in the iNPH group than in the control group.[50,51] He then concluded that the mechanical pressure caused by ventricular enlargement in iNPH patients might induce formation of well-aligned fiber tracts and increased fiber density in the CST, resulting in decreased DK. DKI is able to

depict both the altered microstructure and water molecule movement within neural axons and intra- or extracellular space.

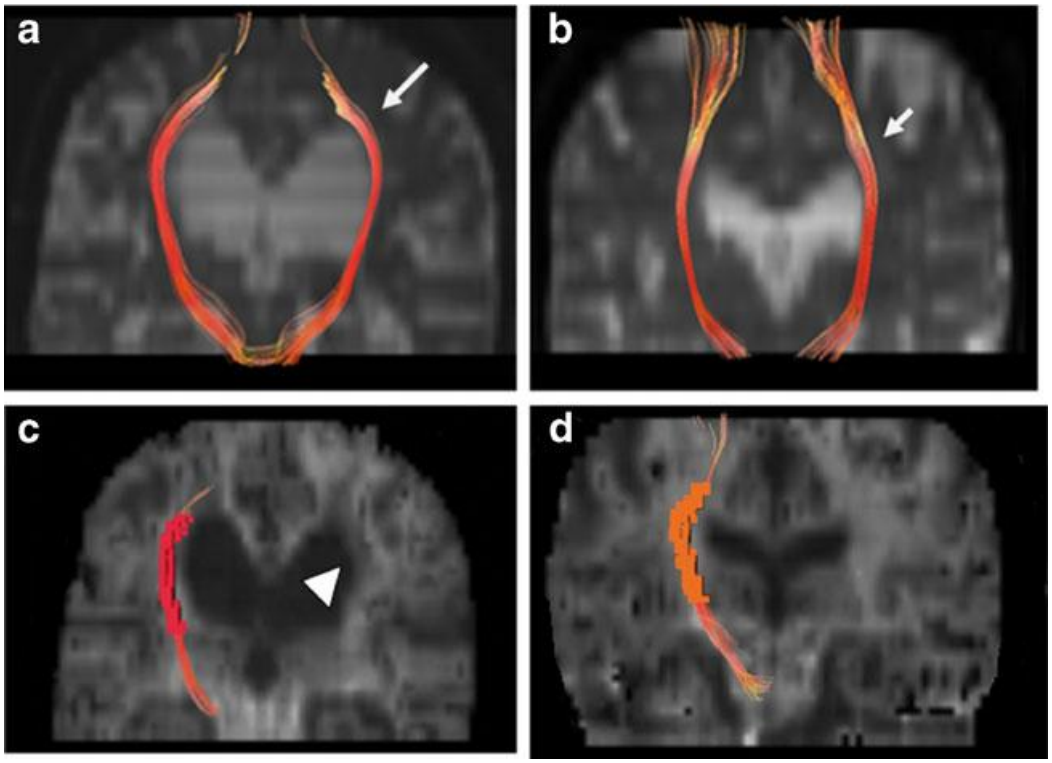

Fig. 3 Anisotropy color-coded MRI tractography of the corticospinal tract (CST) at the level of the lateral ventricle (arrows) (a) in a patient with idiopathic normal pressure hydrocephalus (iNPH) and (b) in a control subject. These tractography on coronal mean kurtosis images are shown without diffusion encoding images (b=0 s/mm2). Diffusional kurtosis images of (c) the iNPH patient and (d) the control subject. The diffusional kurtosis images of the iNPH patient (c) shows the relative low intensity (arrow head) of the periventricular areas compared to control subjects (d) (Nakanishi et al, 2013)

Algin et al conducted a study in 2010 titled MR cisternography: "Is it useful in the diagnosis of normal-pressure hydrocephalus and the selection of "good shunt responders"? [52]. 36 patients with the diagnosis of "probable iNPH" were included in the study group and 15 asymptomatic age-matched individuals were included in the control group. The presence of contrast material in the lateral ventricles for more than 24 hours was accepted as a positive diagnosis of INPH. All of the INPH

patients had remaining contrast material in their lateral ventricles at the 12th and 24th hours, while only 28 (78%) patients had contrast material remaining at the 48th hour after MRC. Only 3 (20%) of the control cases had remaining contrast material in their lateral ventricles at the 24th hour.[53,54] No contrast material was present in the control cases at the 48th hour. The contrast material was found to be significantly more prevalent in the INPH patients at the 24th and the 48th hours compared with the control cases ($P< 0.001$). Shunt placement was performed in 14 INPH patients, and eight improved after shunt placement. All patients (100%) who improved after shunt placement had remaining contrast material in their lateral ventricles at the 24th and at the 48th hours. The sensitivity and specificity of MRC in the prediction of the response to shunt treatment were 100% and 17%, respectively. He concluded that MRC does not use ionizing radiation and is generally a useful procedure to diagnose NPH and to predict a positive response to shunt treatment

Eide et al conducted a study in 2009 titled "Arterial blood pressure vs intracranial pressure in normal pressure hydrocephalus", to characterize the association between arterial blood pressure (ABP) and intracranial pressure (ICP) in idiopathic normal pressure hydrocephalus (iNPH) patients, and its impact on outcome of shunt surgery.[55] It is yet to be concluded if the shunt response in NPH gets affected by the high frequency of arterial hypertension [56], or the high frequency of vascular co-morbidity in NPH [57,58]. The static and pulsatile pressures were averaged over consecutive 6-s intervals; the moving correlations between ICP and ABP (static and pulsatile) were determined during consecutive 4-min periods to explore time-related variations. Results – Neither static nor pulsatile ABP were altered in iNPH shunt responders. Elevated pulsatile ICP, but normal static ICP, was seen in responders. The time-varying correlations of static and of pulsatile pressures

were generally low, and did not differ between shunt responders / non-responders. He concluded that in iNPH shunt responders, static or pulsatile ABP were not altered and only pulsatile ICP was elevated.

TREATMENT

Eide et al conducted a study in 2010 titled "Cerebrospinal fluid pulse pressure (CSFP) amplitude during lumbar infusion in idiopathic normal pressure hydrocephalus can predict response to shunting".[59] The study population consisted of all iNPH patients undergoing both diagnostic lumbar infusion testing and continuous over-night ICP monitoring. Totally 62 iNPH patients were included, 45 of them underwent shunt surgery, in whom 78% were shunt responders. Among the 45 shunted patients, resistance to CSF outflow (Rout) was elevated in 44. The ICP pulse amplitude recorded over-night was elevated in 68% of patients; 92% of these were shunt responders. Elevated CSFP pulse amplitude during lumbar infusion predicted shunt response with sensitivity of 88 and specificity of 60. He concluded that in iNPH patients, shunt response can be anticipated in 9/10 patients with elevated overnight ICP pulse amplitude, while in only 1/10 with low ICP pulse amplitude.[60] The CSFP pulse amplitude during lumbar infusion testing was elevated in patients with elevated over-night ICP pulse amplitude.[61] In particular, measurement of CSFP pulse amplitude during a standardized infusion of 15 ml Ringer over 10 min was useful in predicting response to shunt surgery and can be used as a screening procedure for selection of iNPH patients for shunting.

Johan Virhammar, MD et al. conducted a study in 2018 titled "Increase in callosal angle and decrease in ventricular volume after shunt surgery in patients with idiopathic normal pressure hydrocephalus".[62] It is known that the size of the

ventricles in rapidly developing noncommunicating and posthemorrhagic hydrocephalus is often significantly reduced after shunting but is not so evident in iNPH, and the EI is often unchanged after the shunt surgery.**[63]** The aim of this study was to investigate whether the angle between the lateral ventricles (the callosal angle [CA]) increases and ventricular volume decreases after shunt surgery in patients with iNPH. The CA was larger postoperatively than preoperatively. The volume of the lateral ventricle contralateral to the shunt valve decreased from 73 ml (95% CI 66–80 ml) preoperatively to 63 ml (95% CI 54–72 ml) postoperatively.[64,65] The Evans index was 0.365 preoperatively and 0.358 postoperatively. Postoperative change of CA showed a negative correlation with change of ventricular volume.[66] He concluded that in this sample of patients with iNPH, the CA increased and ventricular volume decreased after shunt surgery.

L. Sakkaa et al in his 2011 article named "Anatomy and physiology of cerebrospinal fluid" explained about cerebrospinal fluid secretion and circulation in adults. [67]

Cerebrospinal fluid secretion in adults:

CSF secretion in adults is about 400 to 600 ml per day. Sixty to seventy-five percent of CSF is produced by the choroid plexuses of the lateral ventricles and the tela choroidea of the third and fourth ventricles. The choroid plexuses consists of granular meningeal protrusions into the ventricular lumen, the epithelial surface of which is continuous with the ependymal of the ventricles. They comprise a tuft of fenestrated capillaries. Choroidal cells present microvilli at their apical pole, are interconnected by tight junctions with a variable distribution according to the site on the ventricular wall.[68]

Cerebrospinal fluid circulation

CSF circulation is a dynamic phenomenon and cerebral homeostasis is regulated by CSF circulation. CSF circulates from the sites of secretion to the sites of absorption according to a unidirectional rostrocaudal flow which is in ventricular cavities and a multidirectional flow in the subarachnoid spaces. CSF flow is pulsatile, corresponding to the systolic pulse wave in choroidal arteries.

CSF produced by the choroid plexuses in the lateral ventricles travels through interventricular foramina to the third ventricle and then to the fourth ventricle through the cerebral aqueduct and finally to the subarachnoid spaces via the median aperture (foramen of Magendie) of the fourth ventricle. In the cranial subarachnoid space, CSF circulates rostrally to the villous sites of absorption or caudally to the spinal subarachnoid space. Experimental studies have demonstrated the existence of a communication between CSF spaces and the adventitia of cerebral arteries: red blood cells injected into CSF spaces in the cat pass through the adventitia of cerebral arteries and are then detected in cervical lymph nodes.

The CSF, partly absorbed by spinal arachnoid villi, circulates rostrally to the cranial subarachnoid space. CSF flow is generated by the systolic pulse wave and rapid respiratory waves.[69]

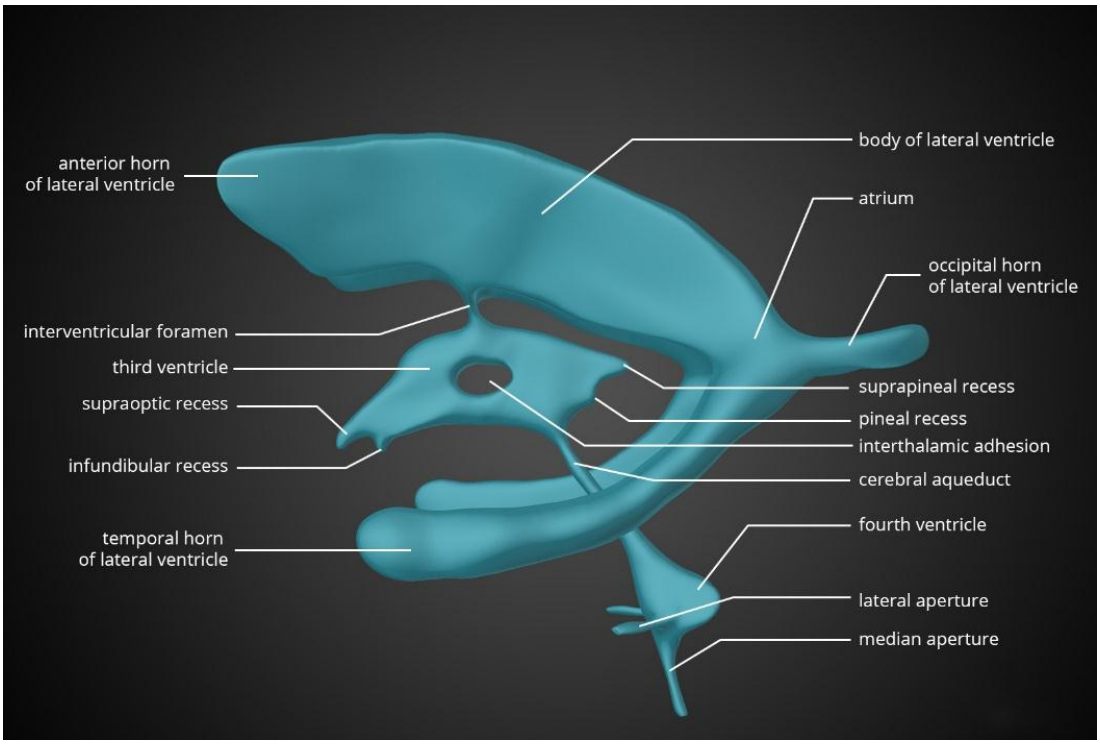

Fig 4. CSF Circulation - Anatomy

MATERIALS & METHODS

MATERIALS AND METHOD

STUDY DESIGN

Prospective Cross sectional

STUDY PERIOD

18 months (Dec 2016 - June 2018)

STUDY SETTING

Department of Radiodiagnosis and Department of Pathology, Sree Mookambika Institute of Medical Science, Kulasekharam, Kanyakumari (District), Tamil Nadu.

STUDY POPULATION

Patients more than 40 years of age, diagnosed with normal pressure hydrocephalus as per consensus criteria and referred to Department of Radiodiagnosis from the department of Neurology

INCLUSION CRITERIA:

- Patients diagnosed with normal pressure hydrocephalus as per consensus criteria and referred to Department of Radiodiagnosis from the department of Neurology, Sree Mookambika Institute of Medical Sciences, Kulasekharam
- Both male and female patients.

EXCLUSION CRITERIA:

- Patients who are having other causes of dementia
- Patients having other significant neurological illness
- Patients not willing to participate

SAMPLE SIZE

Sample size was calculated using the formula

Sample size (n) = 4pq/d2,

Where, p = Prevalence

q = 1 – Prevalence

d = Precision is 20%

P = 73%, Q=27,

Substituting in the sample size formula[70],

$4x73x27/(14.6)^2 = 36.98 \cong 37\%$

Total 40 patients were included in the study.

SAMPLING TECHNIQUE: Convenient sampling technique

STUDY METHOD

After approval by institutional ethical committee, patients of age group more than 40 years of age, of both gender (males and females) diagnosed with normal pressure hydrocephalus as per consensus criteria and referred to Department of Radiodiagnosis from the department of Neurology were enrolled in the study with a written informed consent.

All patients were subjected to Computed Tomography (CT)/ Magnetic Resonance Imaging (MRI)

- After approval of the study protocol by our institutional research & human ethical committee, the patients who fulfilled the inclusion criteria were included in the study. Written informed consent was taken and all the selected patients were explained in detail about the procedure. Magnetic resonance imaging / computed tomography study of brain were performed once.

MAGNETIC RESONANCE IMAGING

- MRI evaluation of brain was done using T1W, T2W, FLAIR, ADC, DWI, GRE,
- 3D CISS sequences. Findings on these sequences was correlated for the study.
- All MRI brain scans are performed on a 1.5T Siemens ESSENZA 16 channel, using head coil.

COMPUTED TOMOGRAPHY

All CT brain scans were performed on 16 slice Siemens somatom spirit.

STATISTICS

Statistical analysis was performed using IBM, SPSS Statistics version 25 (IBM Corp., New York, NY). The test values of continuous variables were expressed as mean ± SD (standard deviation). Means of groups were compared with paired-tests. A p-value < 0.05 was considered statistically significant. Chi-square tests were done for proportions. The Pearson product-moment correlation was used to determine the strength and direction of a linear relationship between two continuous variables and assumptions to run the test were met and there were no outliers. Pearson correlation coefficient, denoted as *r* (i.e., the italic lowercase letter r), measures the strength and direction of a linear relationship between two continuous variables. Its value ranges from -1 for a perfect negative linear relationship to +1 for a perfect positive linear relationship. A value of 0 (zero) indicates no relationship between two variables. Pearson correlation coefficient, $r>0.5$ was considered statistically significant.

RESULTS

RESULTS AND INTERPRETATION

RESULTS

A cross-sectional observational study was done in the department Radiodiagnosis, Sree Mookambika Institute of Medical Sciences, Kulasekharam, Tamil Nadu, India, from July 2016 to April 2018. The trial was approval by the institutional review board and the Ethics committee. A written informed consent was obtained from all participating patients in accordance with the tenets of the declaration of Helsinki.

The number of valid cases with normal pressure hydrocephalus who fulfilled the inclusion criteria were forty. CT and MRI evaluation of brain was done using T1W, T2W, FLAIR, ADC, DWI, GRE, D CISS sequences. The mean age of patients was 65.8±19.2 (range, 42-85 years). Table 1 and Figure 1 shows the age distribution of patients. There were 21(52.5%) males and 19 (47.5%) females, respectively. Table 2 and Figure 2 shows the gender frequency of patients.

The mean Evan index of patients was 0.35±0.04 (range, 0.27-0.44). The distribution of Evans index is shown in Table 3 and Figure 3, respectively. The mean Evans index in males was 0.35±0.04 and in females was 0.34±0.03. Figure 3B compares Evans index between males and females. The difference in Evans index between males and females was not significant (paired t test, P=0.342).

The distribution of Callosal angle of subjects is shown in Table 4 and Figure 4. The mean Callosal angle of patients was 64.7±15.5 (range, 47-95 degrees).

The mean size of third ventricle was 10.4±1.6 (range, 7-13 mm). Table 5 and Figure 5 shows the distribution of the size of third ventricle (mm) in patients.

The mean size of temporal horn was 5.7±2 (range, 2-11 mm). Table 6 and Figure 6 shows the distribution of the size of temporal horn in patients.

The mean cerebrospinal fluid (CSF) pressure was 162±35.7 (range, 100-230 mm of H_2O). Table 7 and Figure 7 shows the distribution of CSF pressure among the patients. The sulci were graded from 0-2. The sulci were normal (0) in 13(32.5%), slight compression (1) in 12 (30%) and definitive compression (3) in 15 (37.5%) patients, respectively. Table 8 and Figure 8 shows the frequency distribution status of sulci in study participants.

Dilation of the Sylvian fissure was graded as normal or narrow (0), mildly moderately enlarged (1) and highly enlarged (2). Ten (25%) had a normal, 13(32.5%) moderately enlarged and 17(42.5%) patients had a highly enlarged sylvian fissure, respectively. Table 9 and Figure 9 shows the status of the sylvian fissure in patients.

Disproportionately enlarged subarachnoid space hydrocephalus (DESH) was graded as absent (0) and present (1), if both narrow sulci at the high convexity and the Sylvian fissure. Amongst the study participants, DESH was noted in 57.5 %, incomplete DESH in 27.5 %, and no DESH in 15 % (Tables 10-12 and Figures 10-12). Figure 12 A compares the three types of DESH.

Focal bulge of the left ventricle was seen in 22 (55%) patients. Table 13 and Figure 13 shows the distribution of left ventricular bulge.

Deep white matter hyperintensities (DWMH) were seen in 36 (90%) participants. These were punctate in 10 (25%), confluent in 13(32.5%) and large confluent in 13 (32.5%), respectively. Table 14 and Figure 14 shows the distribution of hyperintensities in white matter.

Periventricular hyperintensities were absent in 4(10%), increased 18(45%) and irregular, large symmetric with extension to deep white matter in 18 (45%) participants. Table 15 and Figure 15 shows the distribution of periventricular hyperintensities on MRI.

Transport sulci were present in 9 (22.5%) patients. Table 16 and Figure 16 shows the distribution of transport sulci in study participants. None of the participants had aqueduct stenosis (Table 17 and Figure 17).

The CSF tap test was positive in 25 (62.5%) cases. The results of this test are depicted in Table 18 and Figure 18, respectively.

Urinary incontinence was present in 28(70%) cases. Table 19 and Figure 19 shows the distribution of urinary incontinence amongst participants.

Dementia was present in 36 (90%) cases. Table 20 and Figure 20 shows the distribution of dementia in study participants.

Gait ataxia was present in 30 (75%) cases. Figure 21 and Table 21 shows the distribution of Gait ataxia amongst study participants.

Flow voids were absent in 28 (70%) patients. On patient (2.5%) had flow in aqueduct, 5(12.5%) had flow in aqueduct and upper part of 4^{th} ventricle and 6(15%) had flow in caudal part of 4^{th} ventricle. Figure 22 and Table 22 shows the distribution of flow voids in study participants.

The mean CSF pressure in patients with gait ataxia was 159.6±32 mm of H_2O. The mean CSF pressure in patients without CSF pressure was 175±45 mm of H_2O. Figure 23 and Table 23 shows CSF pressure in patients with and without ataxia.

Correlation analysis was done between MRI and clinical parameters to diagnose normal pressure hydrocephalus. There was a significant correlation ($P<0.001$) between Evans index and the size of the third ventricle (Pearson's correlation coefficient, r=0.525). Table 24 and Figure 24 shows the correlation between these parameters.

Callosal angle correlated significantly (P=0.03) with focal left ventricle bulge (Pearson's correlation coefficient, r= 0.338). Table 25 and Figure 25 shows the correlation between these parameters.

Evans index correlated significantly (P=0.03) with cerebrospinal fluid pressure (Pearson's correlation coefficient, r= 0.335). Table 26 and Figure 26 shows the correlation between these parameters. Evans index also correlated significantly (P=0.001) with size of temporal horn (Pearson's correlation coefficient, r= 0.522). Table 27 and Figure 27 shows the correlation between these parameters.

The correlation between callosal angle and maximal diameter of temporal horn (Pearson's correlation coefficient, r=0.06) was not significant (P=0.714). Table 28 and Figure 28 shows the correlation between these parameters.

Deep white matter hyperintensities correlated significantly (P=0.003) with periventricular hyperintensities (Pearson's correlation coefficient, r= 0.459). Table 29 and Figure 29 shows the correlation between the two hyperintensities.

Complete Disproportionately enlarged subarachnoid space hydrocephalus (DESH) correlated significantly (P=0.05) with maximum diameter of temporal horn ((Pearson's correlation coefficient, r= 0.317). Table 30 and Figure 30 shows the correlation between the two parameters.

There was a significant inverse correlation (P=0.01) between positive CSF tap test and gait ataxia (Pearson's correlation coefficient, r= -0.388). Figure 31 shows the correlation between these parameters.

Figure 5

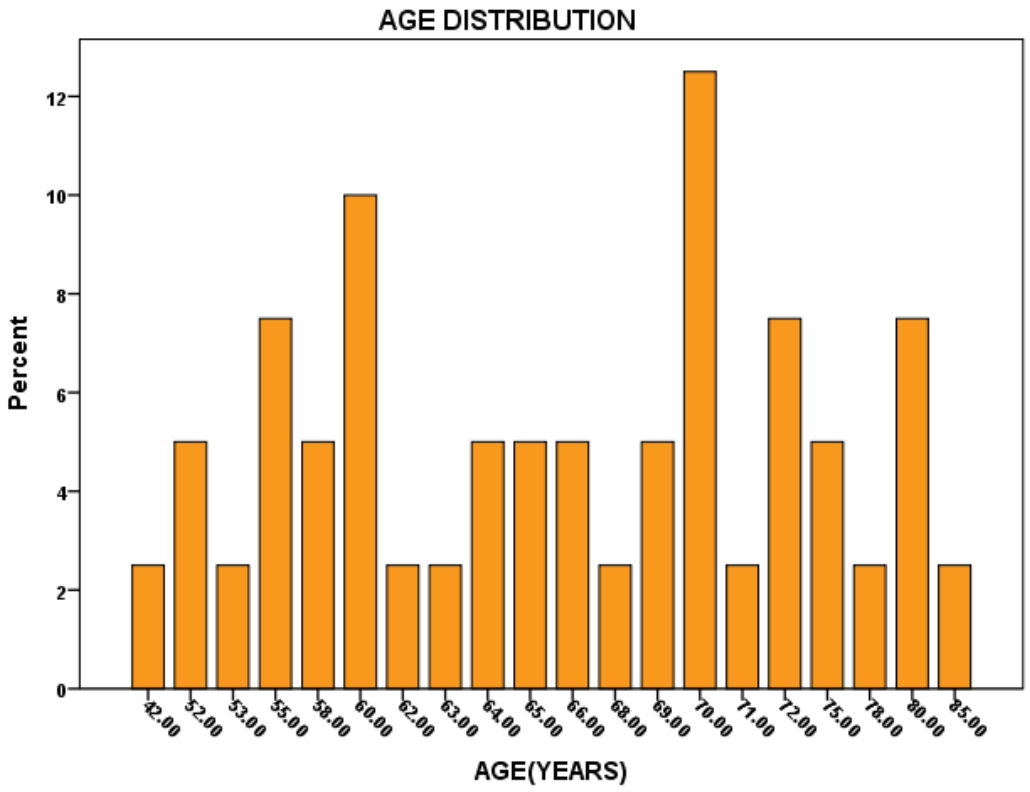

Table 1

AGE DISTRIBUTION

Age (years)	Frequency	Percent	Valid Percent	Cumulative Percent
42	1	2.5	2.5	2.5
52	2	5	5	7.5
53	1	2.5	2.5	10
55	3	7.5	7.5	17.5
58	2	5	5	22.5
60	4	10	10	32.5
62	1	2.5	2.5	35
63	1	2.5	2.5	37.5
64	2	5	5	42.5
65	2	5	5	47.5
66	2	5	5	52.5
68	1	2.5	2.5	55
69	2	5	5	60
70	5	12.5	12.5	72.5
71	1	2.5	2.5	75
72	3	7.5	7.5	82.5
75	2	5	5	87.5
78	1	2.5	2.5	90
80	3	7.5	7.5	97.5
85	1	2.5	2.5	100
Total	40	100	100	

Table 2

GENDER DISTRIBUTION

	Frequency	Percent	Valid Percent	Cumulative Percent
Male	21	52.5	52.5	52.5
Female	19	47.5	47.5	100
Total	40	100	100	

Figure 6

GENDER DISTRIBUTION

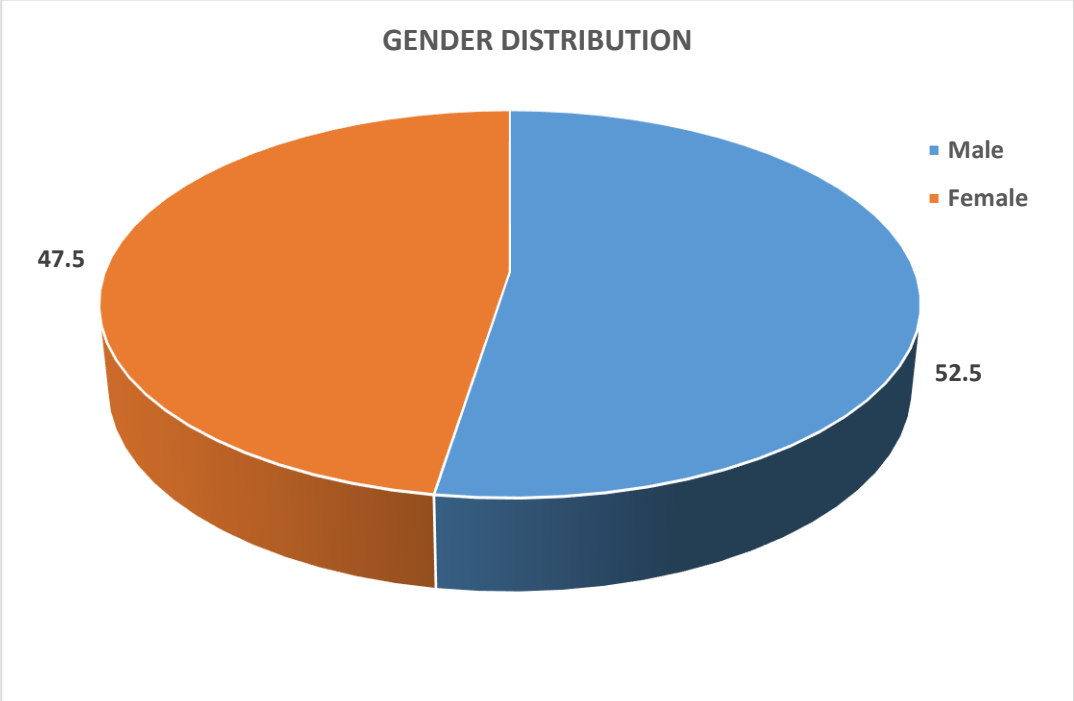

Table 3

EVANS INDEX

	Frequency	Percent	Valid Percent	Cumulative Percent
0.27	1	2.5	2.5	2.5
0.28	1	2.5	2.5	5
0.29	2	5	5	10
0.3	2	5	5	15
0.31	1	2.5	2.5	17.5
0.32	3	7.5	7.5	25
0.33	4	10	10	35
0.34	2	5	5	40
0.35	4	10	10	50
0.36	4	10	10	60
0.37	4	10	10	70
0.38	4	10	10	80
0.39	3	7.5	7.5	87.5
0.4	3	7.5	7.5	95
0.42	1	2.5	2.5	97.5
0.44	1	2.5	2.5	100
Total	40	100	100	

Figure 7

EVANS INDEX

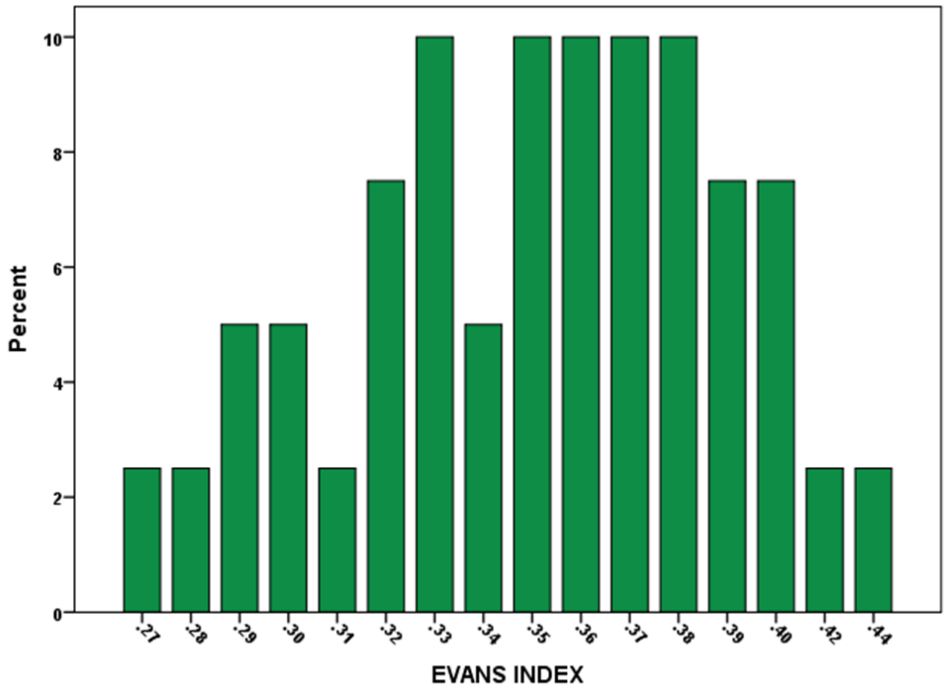

Figure 8

EVANS INDEX WITH GENDER CORELATION

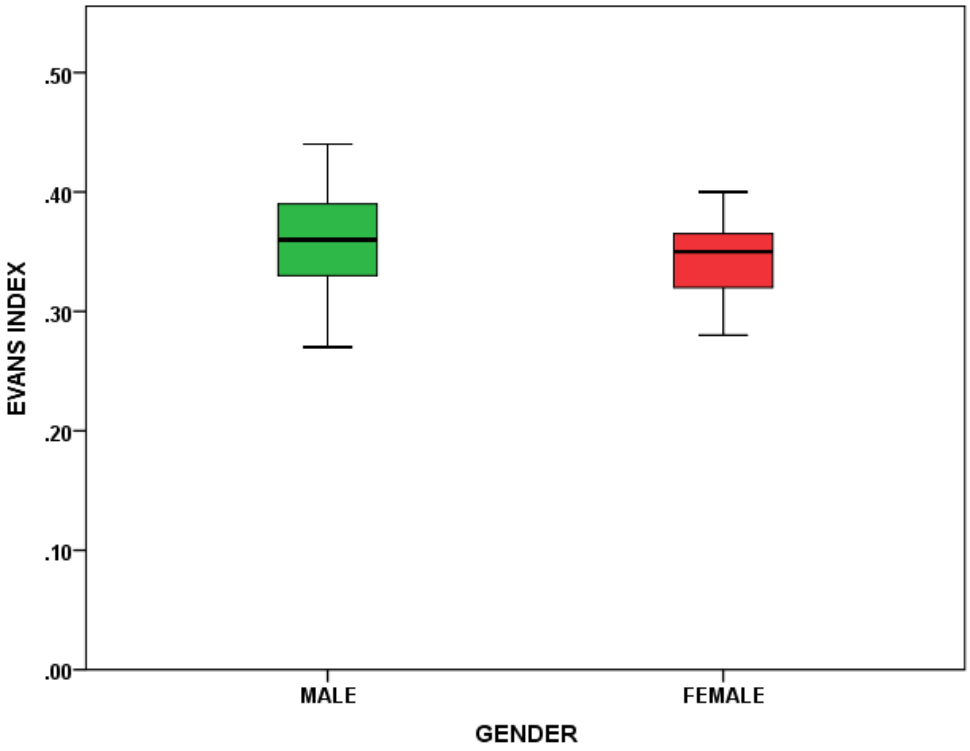

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Table 4

CALLOSAL ANGLE

	Frequency	Percent	Valid Percent	Cumulative Percent
47	1	2.5	2.5	2.5
48	2	5	5	7.5
49	1	2.5	2.5	10
50	1	2.5	2.5	12.5
52	2	5	5	17.5
54	1	2.5	2.5	20
55	3	7.5	7.5	27.5
56	2	5	5	32.5
58	2	5	5	37.5
62	2	5	5	42.5
63	2	5	5	47.5
65	1	2.5	2.5	50
66	1	2.5	2.5	52.5
67	1	2.5	2.5	55
68	2	5	5	60
69	2	5	5	65
70	1	2.5	2.5	67.5
72	1	2.5	2.5	70
75	1	2.5	2.5	72.5
76	3	7.5	7.5	80
77	3	7.5	7.5	87.5
82	1	2.5	2.5	90
83	1	2.5	2.5	92.5
89	2	5	5	97.5
95	1	2.5	2.5	100
Total	40	100	100	

Figure 9

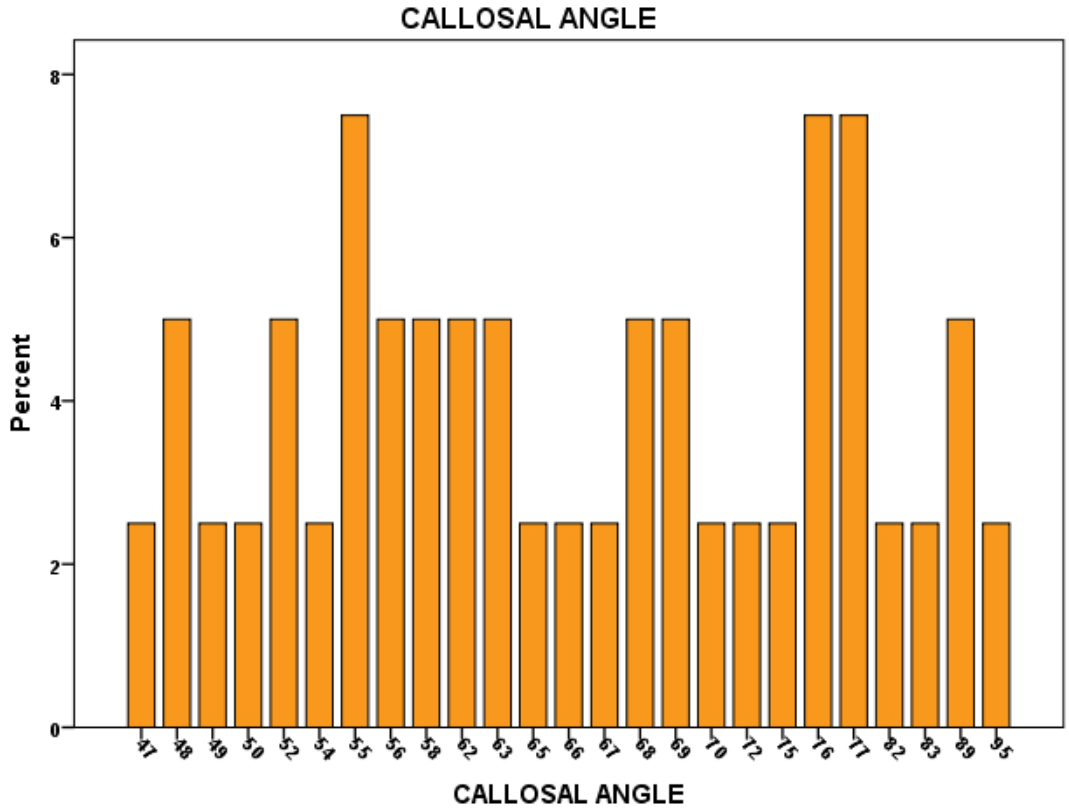
CALLOSAL ANGLE
Percent
0
2
4
6
8
41
48
49
50
52
54
55
56
58
62
63
65
66
67
68
69
70
72
75
76
77
82
83
89
95
CALLOSAL ANGLE

Table 5

SIZE OF THIRD VENTRICLE

Size (mm)	Frequency	Percent	Valid Percent	Cumulative Percent
7	1	2.5	2.5	2.5
8	4	10	10	12.5
9	1	2.5	2.5	15
9	5	12.5	12.5	27.5
10	2	5	5	32.5
10	1	2.5	2.5	35
10	4	10	10	45
10	1	2.5	2.5	47.5
10	1	2.5	2.5	50
11	1	2.5	2.5	52.5
11	9	22.5	22.5	75
11	1	2.5	2.5	77.5
12	4	10	10	87.5
13	5	12.5	12.5	100
Total	40	100	100	

Figure 10

SIZE OF THIRD VENTRICLE (mm)

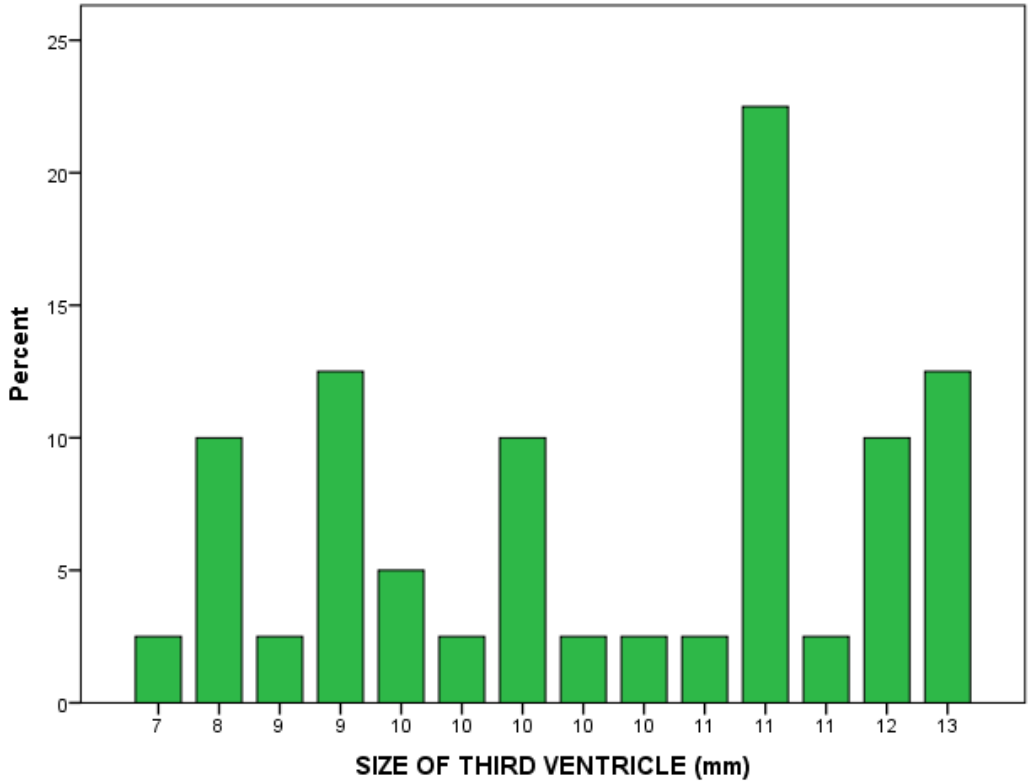

Table 6

SIZE OF TEMPORAL HORN

Size (mm)	Frequency	Percent	Valid Percent	Cumulative Percent
2	1	2.5	2.5	2.5
10.1-15	2	5	5	7.5
3	2	5	5	12.5
3	1	2.5	2.5	15
4	1	2.5	2.5	17.5
4	1	2.5	2.5	20
4	2	5	5	25
4	2	5	5	30
5	1	2.5	2.5	32.5
5	1	2.5	2.5	35
5	2	5	5	40
5	4	10	10	50
5	1	2.5	2.5	52.5
6	1	2.5	2.5	55
6	1	2.5	2.5	57.5
6	3	7.5	7.5	65
7	1	2.5	2.5	67.5
7	4	10	10	77.5
8	1	2.5	2.5	80
8	1	2.5	2.5	82.5
8	3	7.5	7.5	90
8	1	2.5	2.5	92.5
9	1	2.5	2.5	95
10	1	2.5	2.5	97.5
11	1	2.5	2.5	100
Total	40	100	100	

Figure 11

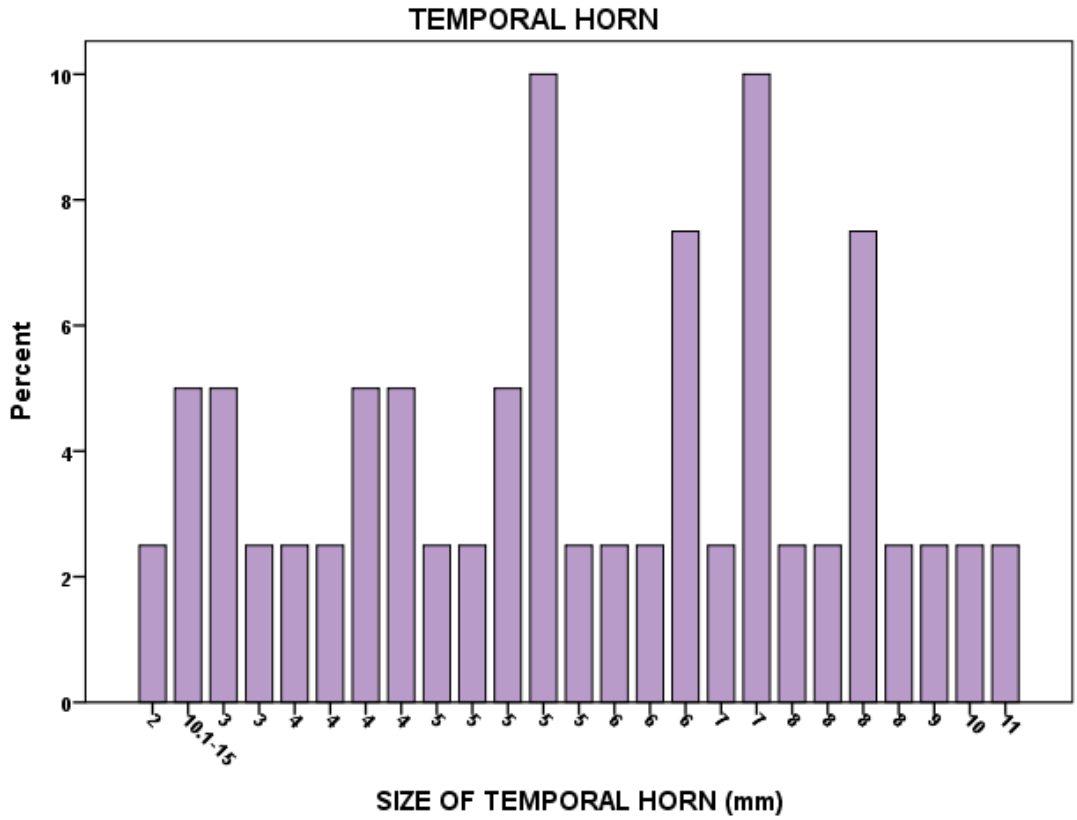
TEMPORAL HORN
Percent
0
2
4
6
8
10
2
10.1-15
3
3
4
4
4
4
5
5
5
5
5
5
6
6
6
7
7
8
8
8
8
9
10
11
SIZE OF TEMPORAL HORN (mm)

Table 7

CEREBROSPINAL FLUID PRESSURE

Pressure (mm of H_2O)	**Frequency**	**Percent**	**Valid Percent**	**Cumulative Percent**
100	1	2.5	2.5	2.5
110	4	10	10	12.5
120	3	7.5	7.5	20
130	3	7.5	7.5	27.5
140	3	7.5	7.5	35
150	3	7.5	7.5	42.5
160	3	7.5	7.5	50
170	4	10	10	60
180	5	12.5	12.5	72.5
190	4	10	10	82.5
200	1	2.5	2.5	85
210	3	7.5	7.5	92.5
220	2	5	5	97.5
230	1	2.5	2.5	100
Total	40	100	100	

Figure 12

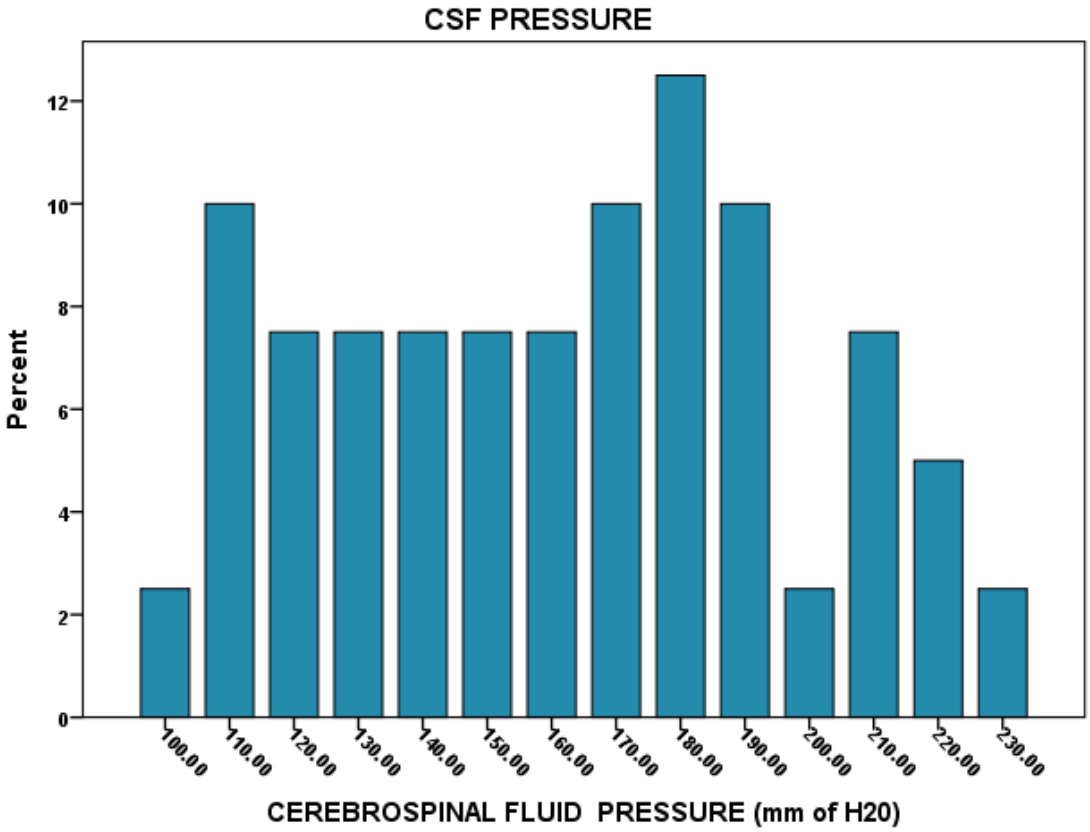
CSF PRESSURE
Percent
0
2
4
6
8
10
12
100.00
110.00
120.00
130.00
140.00
150.00
160.00
170.00
180.00
190.00
200.00
210.00
220.00
230.00
CEREBROSPINAL FLUID PRESSURE (mm of H20)

Table 8

NARROW SULCI

Findings	Frequency	Percent	Valid Percent	Cumulative Percent
Normal	13	32.5	32.5	32.5
Slight Compression	12	30	30	62.5
Definitive Compression	15	37.5	37.5	100
Total	40	100	100	

Figure 13

NARROW SULCI

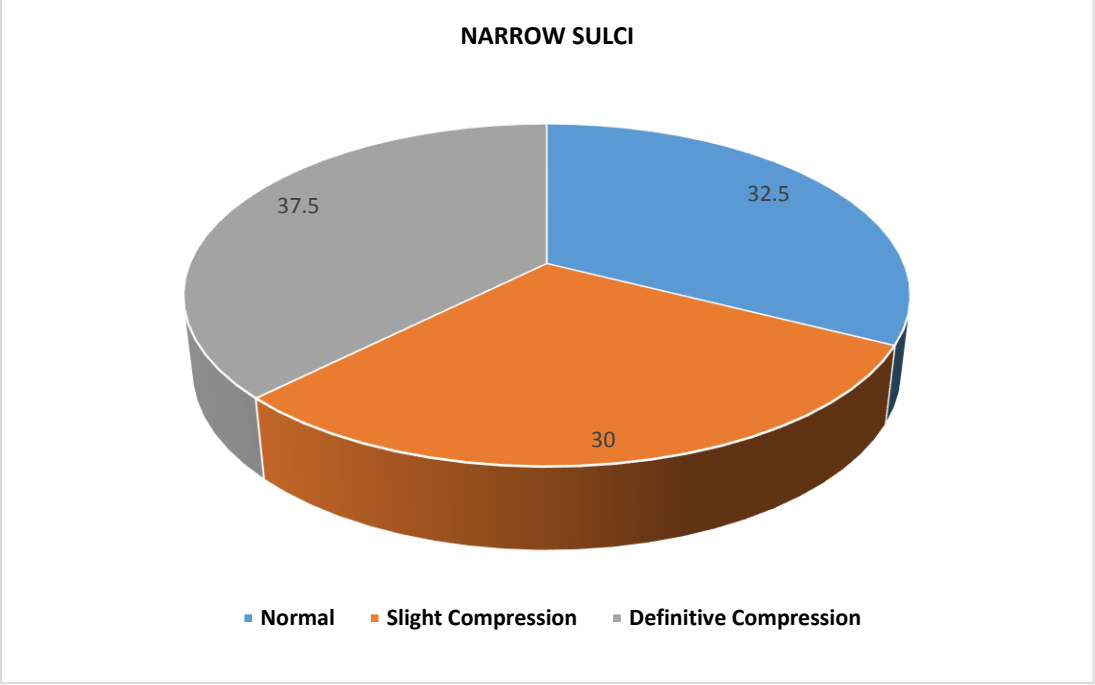

Table 9

SYLVIAN FISSURE DILATION

Findings	Frequency	Percent	Valid Percent	Cumulative Percent
Normal	10	25	25	25
Mildly Enlarged	13	32.5	32.5	57.5
Highly enlarged	17	42.5	42.5	100
Total	40	100	100	

Figure 14

SYLVIAN FISSURE DILATATION

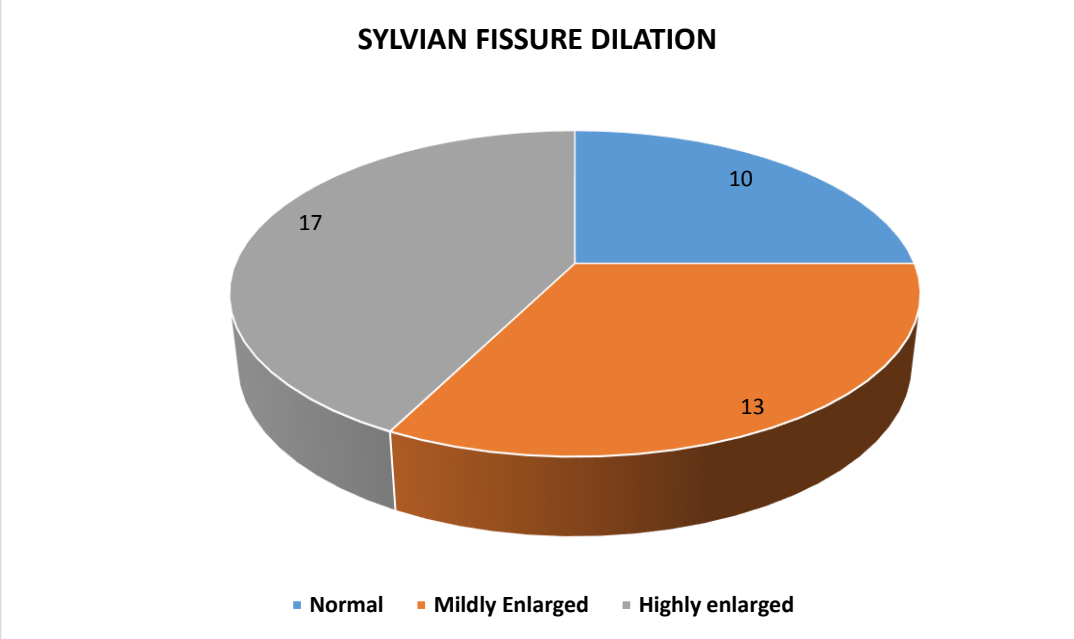

Table 10

COMPLETE DISPROPORTIONATELY

ENLARGED SUBARACHNOID SPACE HYDROCEPHALUS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	16	40.0	41.0	41.0
	Present	23	57.5	59.0	100.0
	Total	39	97.5	100.0	
Missing	System	1	2.5		
Total		40	100.0		

Fig 15

COMPLETE DESH

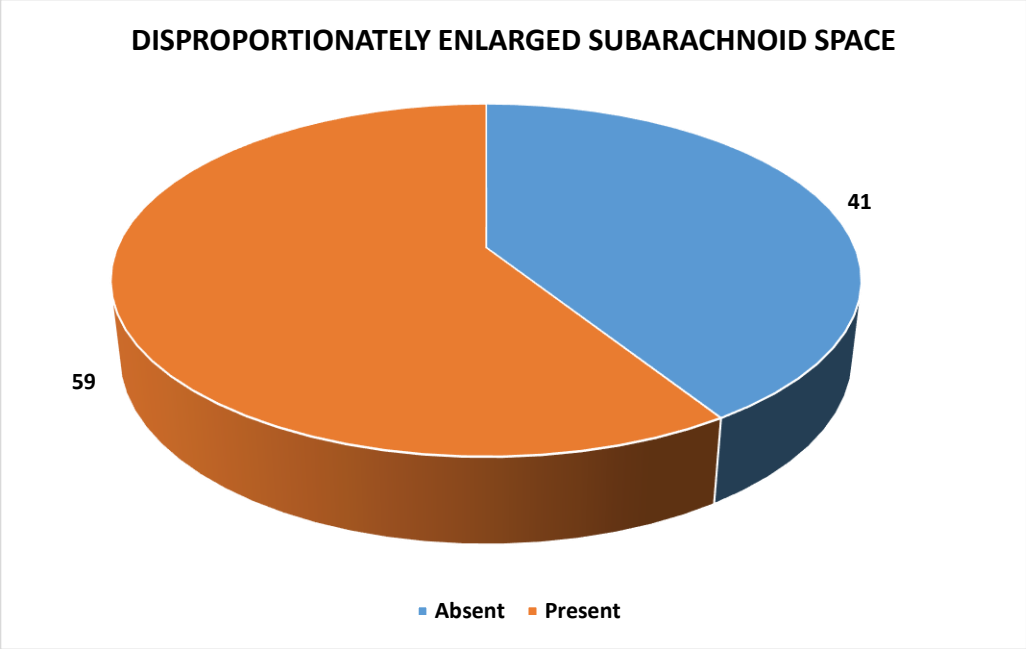

Table 11

INCOMPLETE DISPROPORTIONATELY ENLARGED SUBARACHNOID SPACE HYDROCEPHALUS

	Frequency	Percent	Valid Percent	Cumulative Percent
Absent	29	72.5	72.5	72.5
Present	11	27.5	27.5	100.0
Total	40	100.0	100.0	

Figure 16

INCOMPLETE DESH

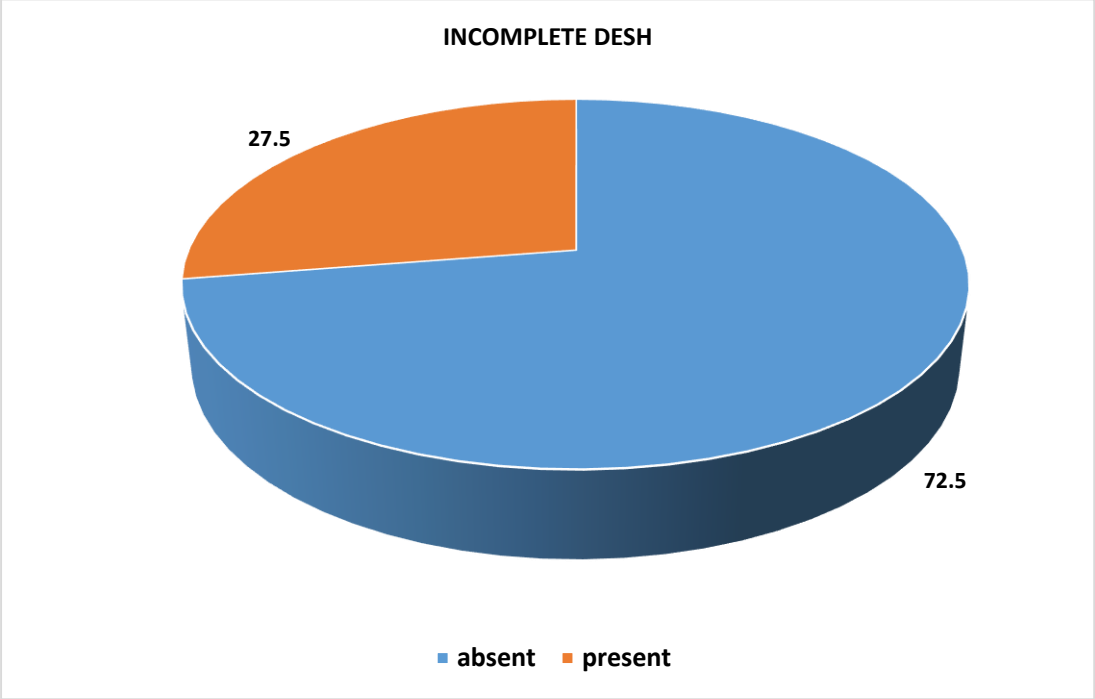

Table 12

NON DISPROPORTIONATELY

ENLARGED SUBARACHNOID SPACE HYDROCEPHALUS

	Frequency	Percent	Valid Percent	Cumulative Percent
Absent	34	85.0	85.0	85.0
Present	6	15.0	15.0	100.0
Total	40	100.0	100.0	

Figure 17

NON DESH

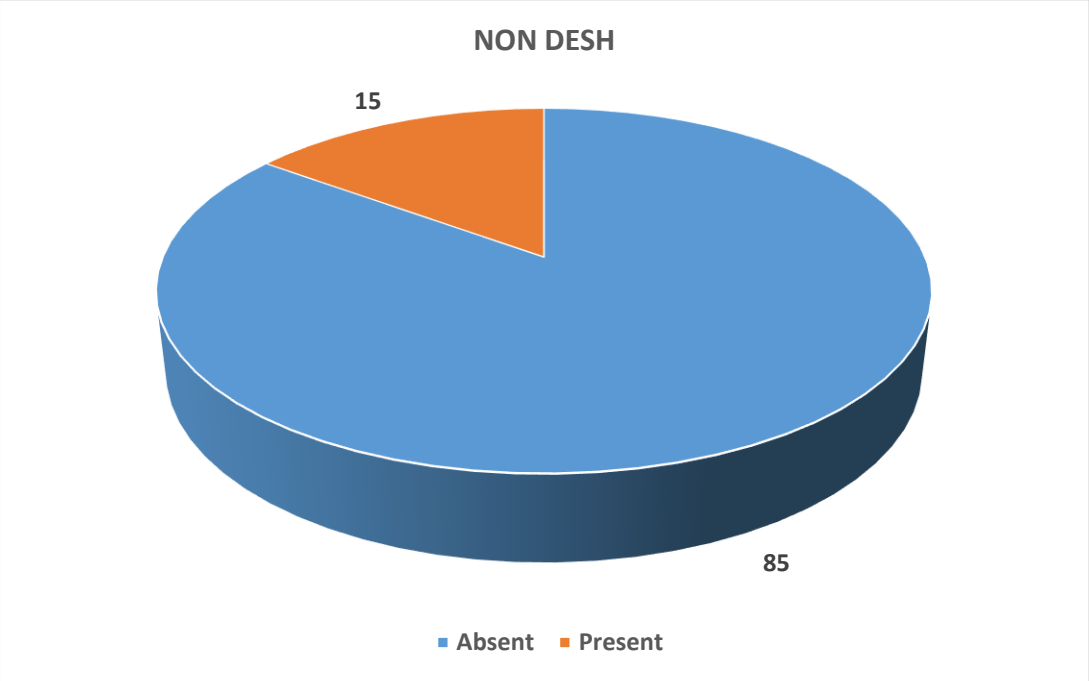

Figure 18

PREVALENCE OF DESH TYPES

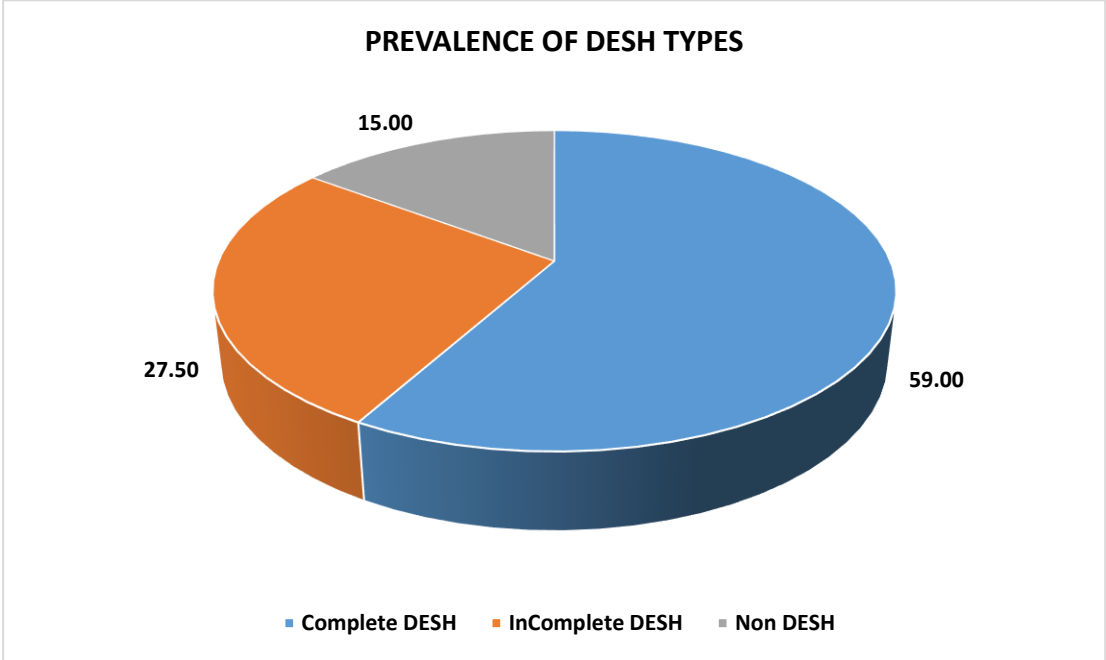

Table 13

FOCAL LEFT VENTRICLE BULGE

	Frequency	Percent	Valid Percent	Cumulative Percent
Absent	18	45.0	45.0	45.0
Present	22	55.0	55.0	100.0
Total	40	100.0	100.0	

Figure 19

FOCAL LV BULGE

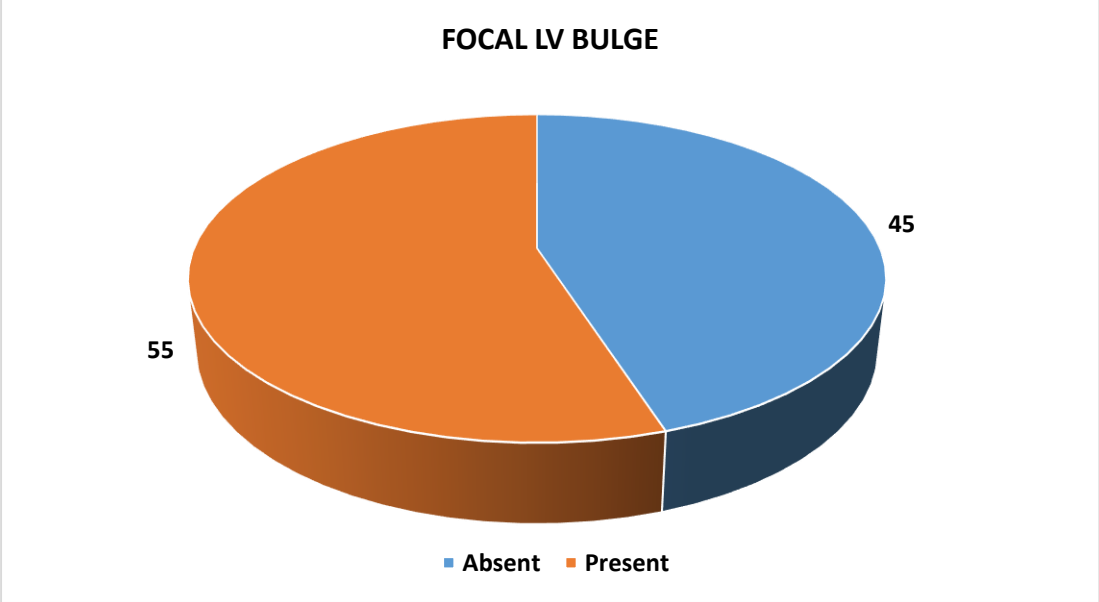

Table 14

DEEP WHITE MATTER HYPERINTENSITIES

	Frequency	Percent	Valid Percent	Cumulative Percent
No lesions	4	10.0	10.0	10.0
Punctate foci	10	25.0	25.0	35.0
Confluent	13	32.5	32.5	67.5
Large confluent	13	32.5	32.5	100.0
Total	40	100.0	100.0	

Figure 20

DEEP WHITE MATTER HYPERINTENSITIES

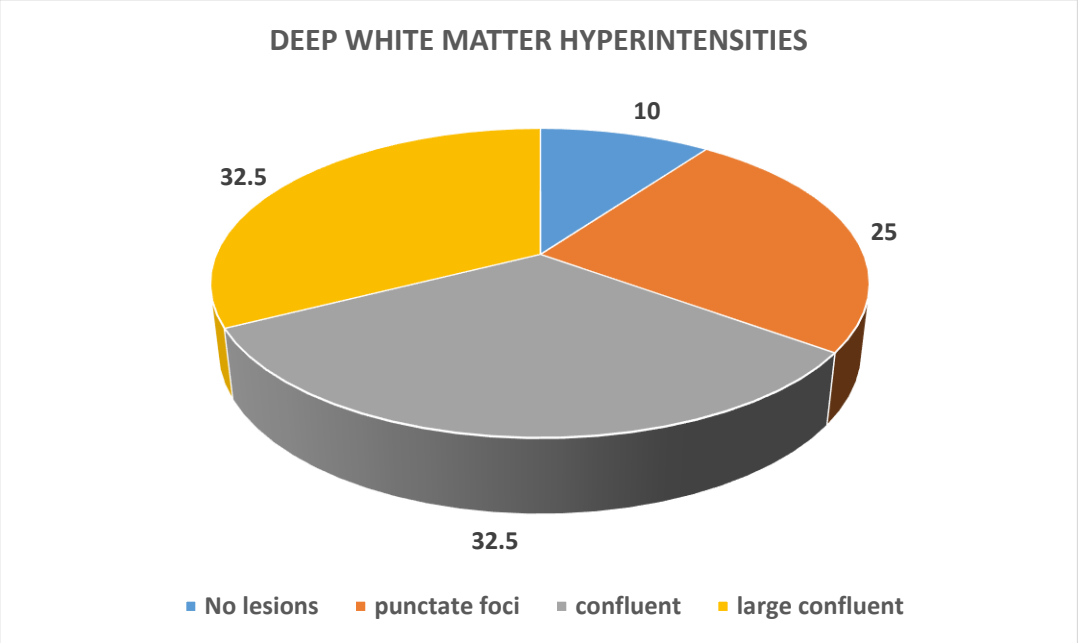

Table 15

PERIVENTRICULAR HYPERINTENSITIES

	Frequency	Percent	Valid Percent	Cumulative Percent
Absent	4	10.0	10.0	10.0
increased	18	45.0	45.0	55.0
irregular large	18	45.0	45.0	100.0
Total	40	100.0	100.0	

Figure 21

PERIVENTRICULAR HYPERINTENSITIES

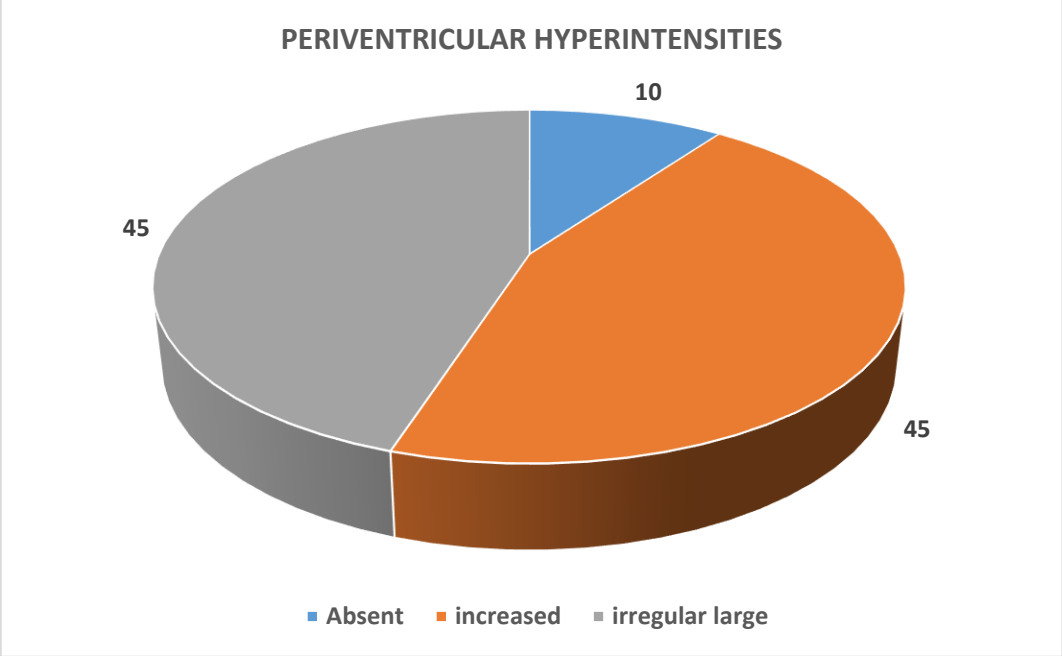

Table 16

TRANSPORT SULCI

	Frequency	Percent	Valid Percent	Cumulative Percent
absent	31	77.5	77.5	77.5
present	9	22.5	22.5	100.0
Total	40	100.0	100.0	

Figure 22

TRANSPORT SULCI

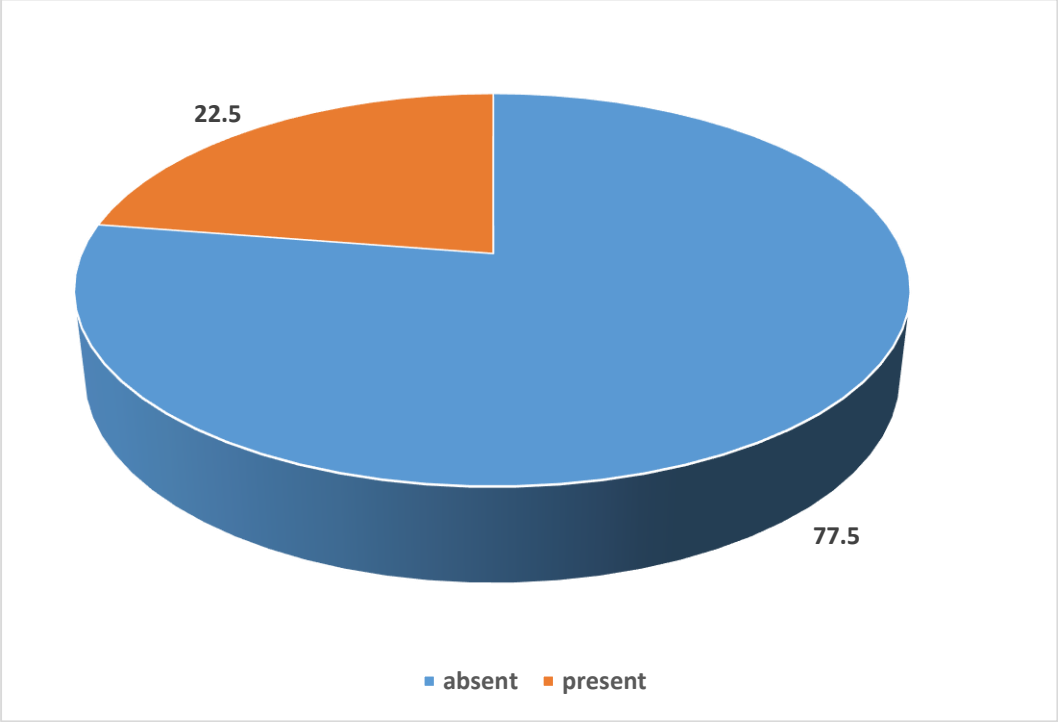

Table 17

AQUEDUCT STENOSIS

			Frequency	Percent	Valid Percent	Cumulative Percent
Valid		Absent	40	100.0	100.0	100.0

Figure 23

AQUEDUCTAL STENOSIS

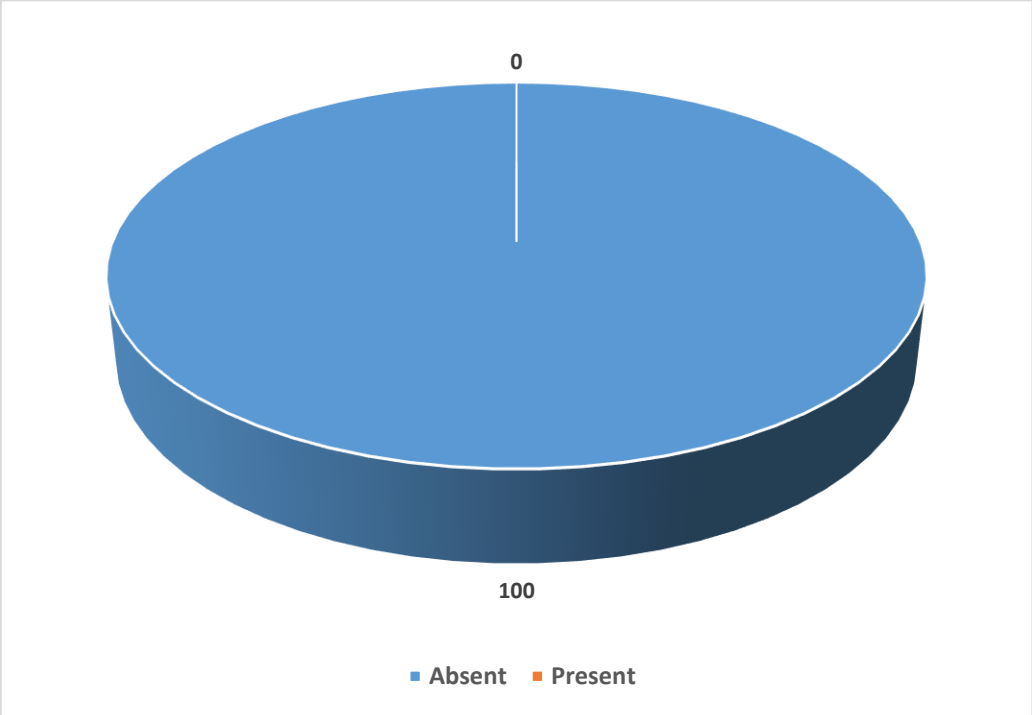

Table 18

CSF TAP TEST

	Frequency	Percent	Valid Percent	Cumulative Percent
Present	25	62.5	62.5	62.5
Absent	15	37.5	37.5	100.0
Total	40	100.0	100.0	

Figure 24

CSF TAP TEST

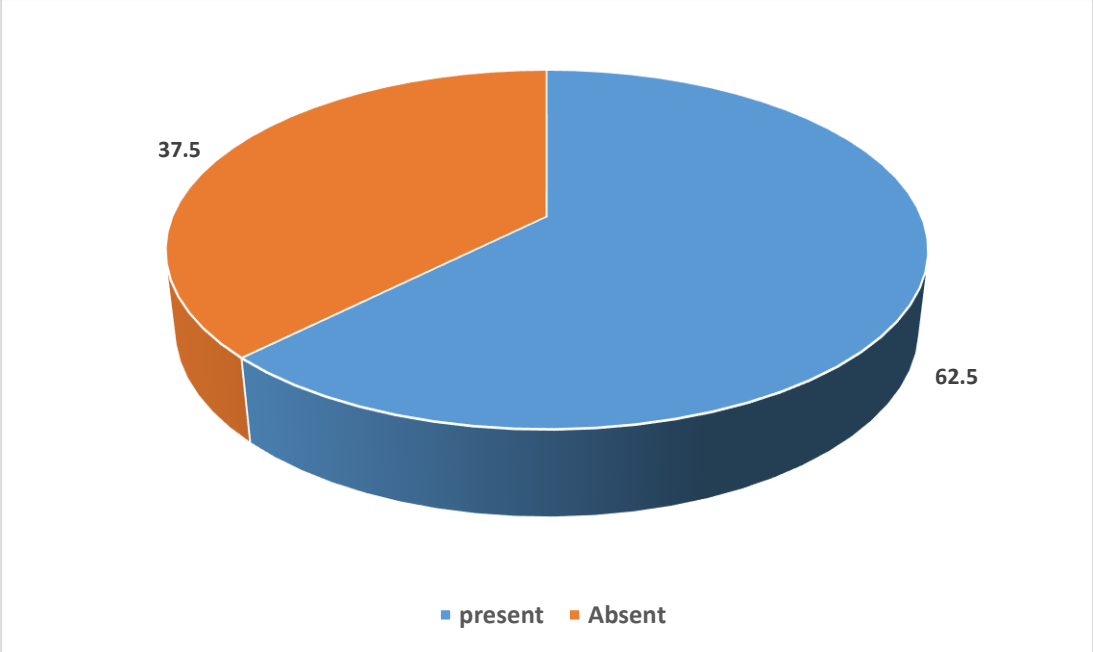

Table 19

URINARY INCONTINENCE

	Frequency	Percent	Valid Percent	Cumulative Percent
Absent	12	30.0	30.0	30.0
present	28	70.0	70.0	100.0
Total	40	100.0	100.0	

Figure 25

URINARY INCONTINENCE

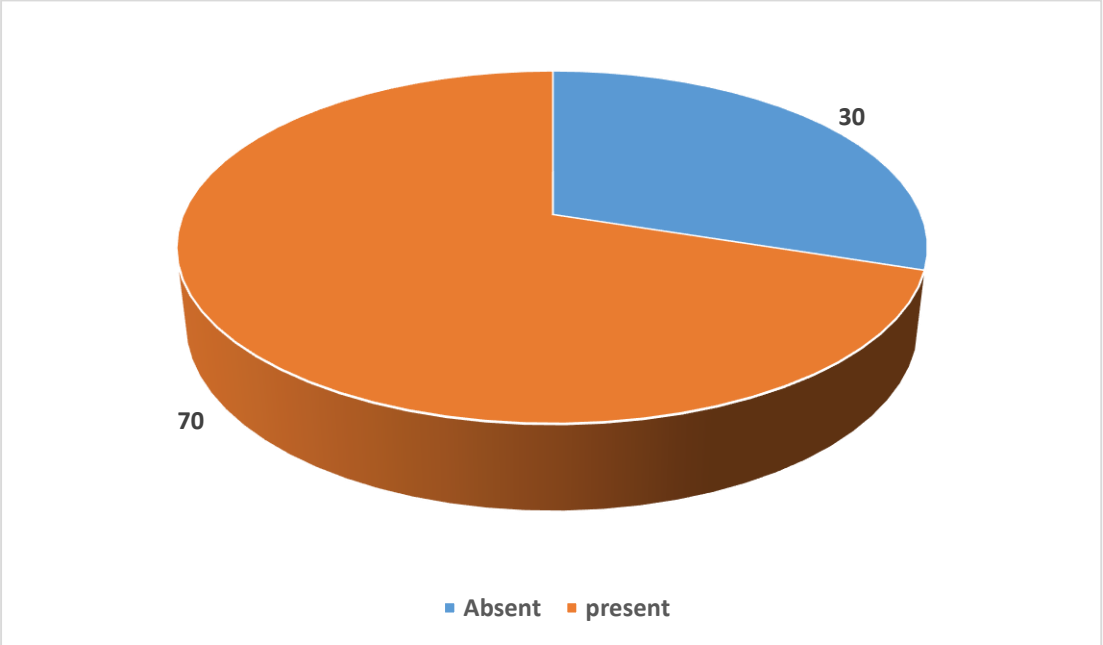

Table 20

DEMENTIA

	Frequency	Percent	Valid Percent	Cumulative Percent
Absent	4	10.0	10.0	10.0
Present	36	90.0	90.0	100.0
Total	40	100.0	100.0	

Figure 26

DEMENTIA

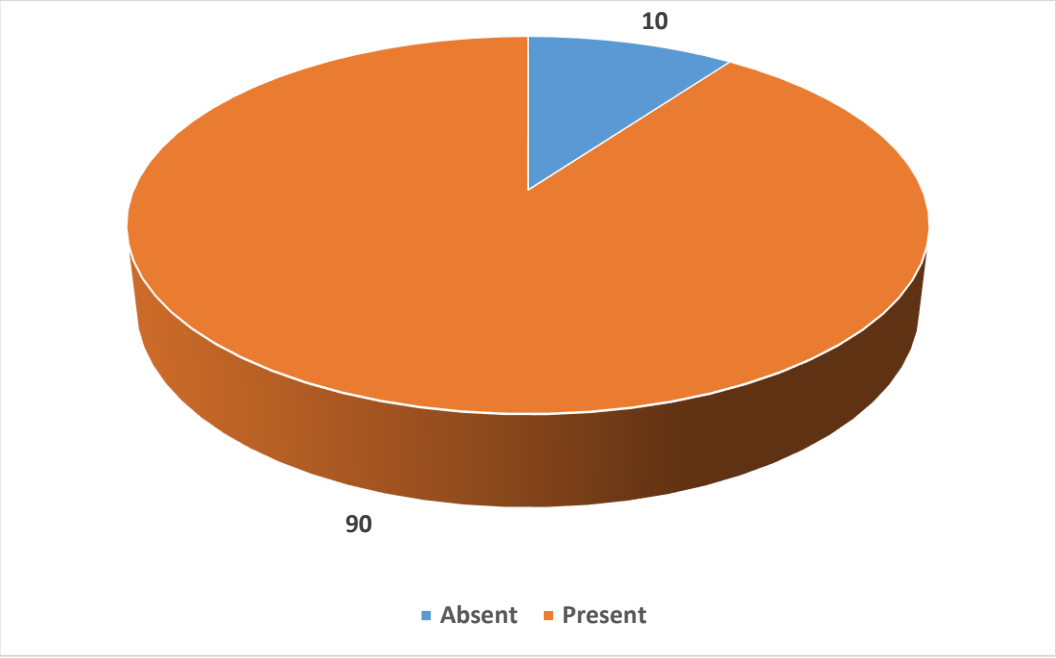

Table 21

GAIT ATAXIA

	Frequency	Percent	Valid Percent	Cumulative Percent
Absent	10	25.0	25.0	25.0
Present	30	75.0	75.0	100.0
Total	40	100.0	100.0	

Figure 27

GAIT ATAXIA

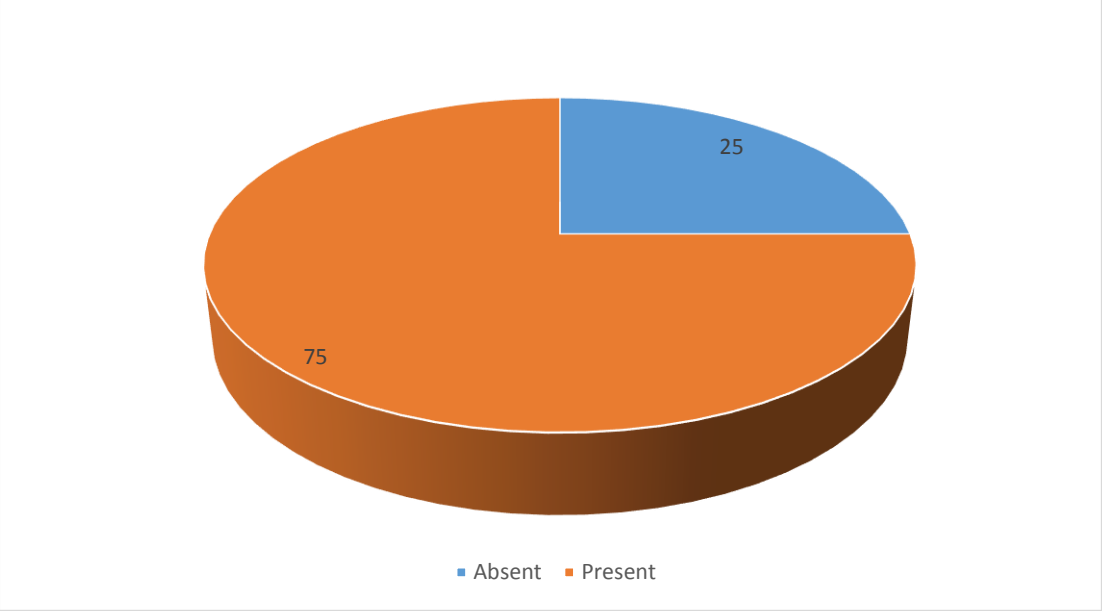

Table 22

FLOW VOIDS

	Frequency	Percent	Valid Percent	Cumulative Percent
Absent	28	70.0	70.0	70.0
Flow in Aqueduct	1	2.5	2.5	72.5
Aqueduct and 4th ventricle upper part	5	12.5	12.5	85.0
4th Ventricle caudal part	6	15.0	15.0	100.0
Total	40	100.0	100.0	

Figure 28

FLOW VOIDS

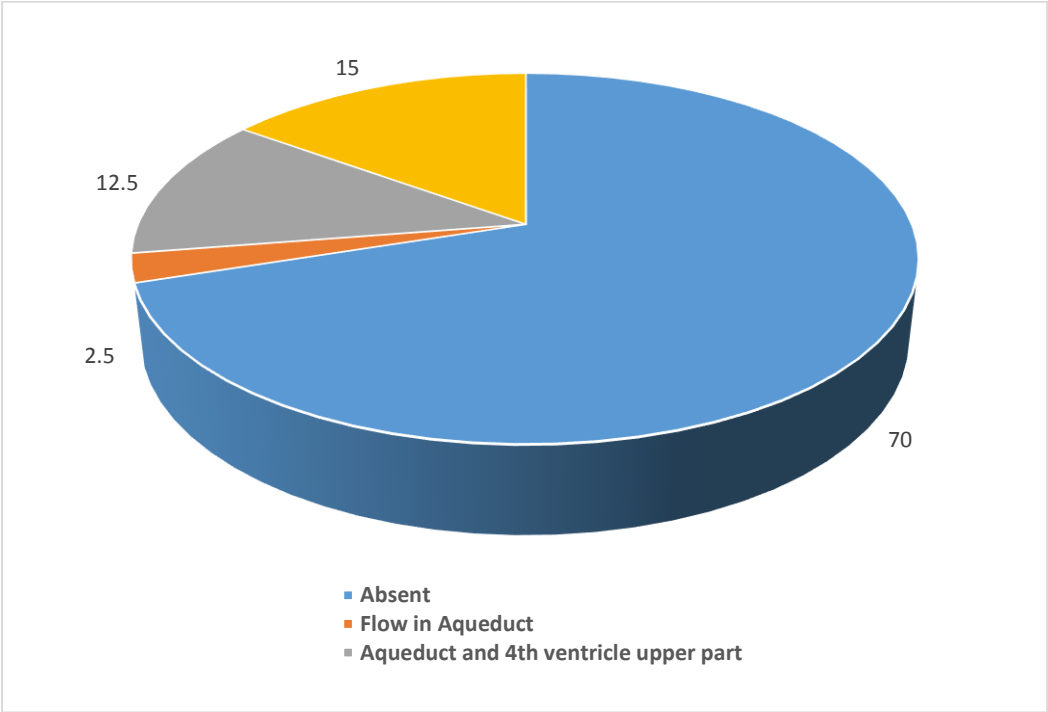

TABLE 23

CEREBROSPINAL FLUID PRESSURE

Group	Mean	N	Std. Deviation
Gait Ataxia Absent	175.0000	12	45.42726
Gait Ataxia Present	159.6429	28	32.02801

Figure 29

CORRELATION BETWEEN GAIT ATAXIA AND MEAN CSF PRESSURE.

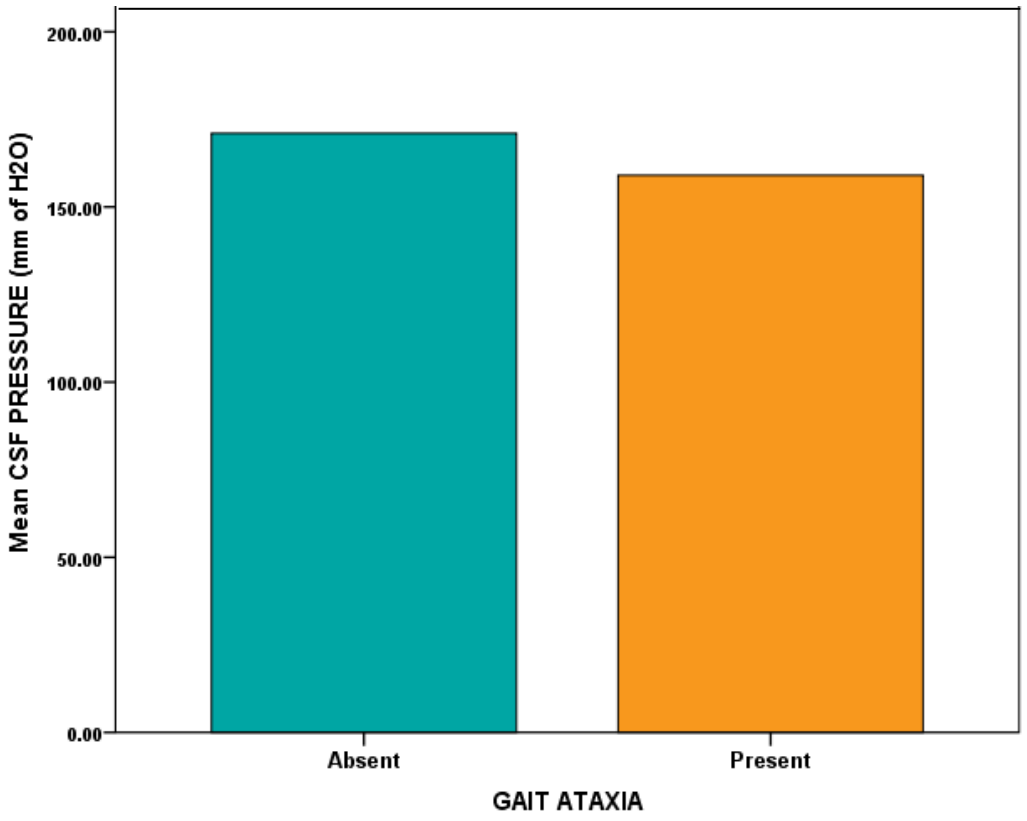

Table 24

PEARSON'S CORRELATION ANALYSIS

		EVANS INDEX	THIRD VENTRICLE
EVANS INDEX	Pearson Correlation	1	.525**
	Sig. (2-tailed)		.001
	N	40	40
THIRD VENTRICLE	Pearson Correlation	.525**	1
	Sig. (2-tailed)	.001	
	N	40	40

**. Correlation is significant at the 0.01 level (2-tailed).

Figure 30

CORRELATION BETWEEN SIZE OF THE THIRD VENTRICLE AND EVANS INDEX.

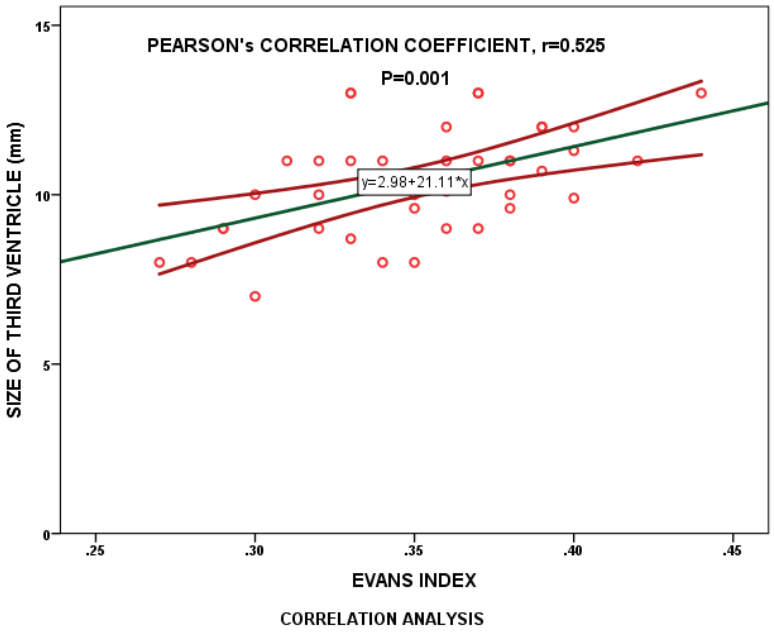

Table 25

PEARSON's CORRELATION ANALYSIS

		CALLOSAL ANGLE	FOCAL LV BULGE
CALLOSAL ANGLE	Pearson Correlation	1	.338*
	Sig. (2-tailed)		.033
	N	40	40
FOCAL LV BULGE	Pearson Correlation	.338*	1
	Sig. (2-tailed)	.033	
	N	40	40

*. Correlation is significant at the 0.05 level (2-tailed).

Figure 31

CORRELATION BETWEEN FOCAL BULGE OF LEFT VENTRICLE AND CALLOSAL ANGLE

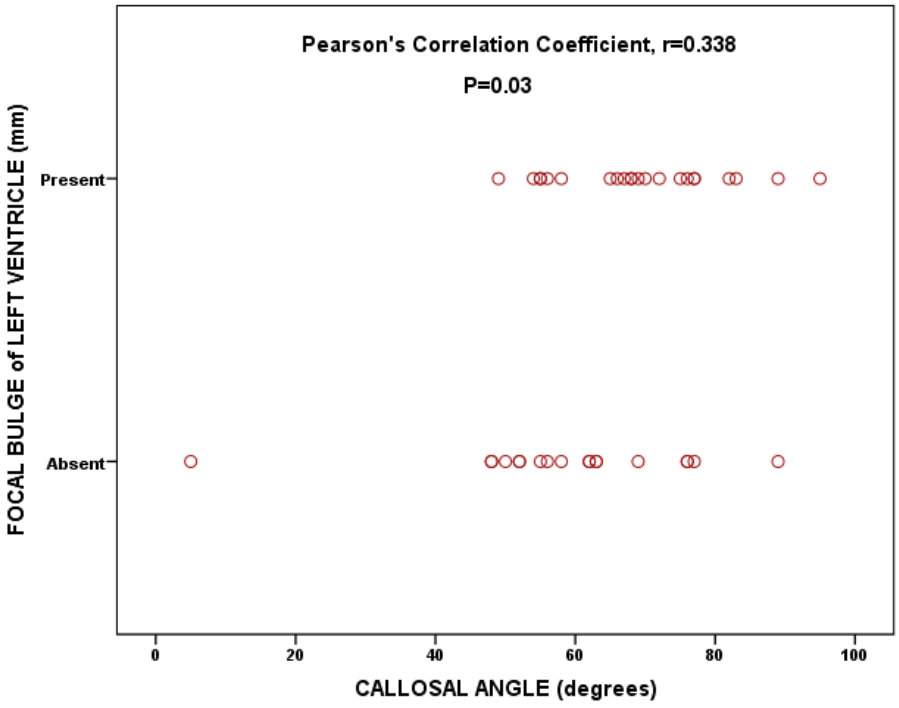

Table 26

CORRELATIONS BETWEEN DIFFERENT PARAMETERS

		CSF pressure	Evans index	Callosal angle	Third ventricle	Temporal horn
CSF Pressure	Pearson Correlation	1	-.335*	-.008	-.150	-.086
	Sig. (2-tailed)		.035	.963	.354	.599
	N	40	40	40	40	40
Evans Index	Pearson Correlation	-.335*	1	-.171	.525**	.522**
	Sig. (2-tailed)	.035		.291	.001	.001
	N	40	40	40	40	40
Callosal Angle	Pearson Correlation	-.008	-.171	1	-.088	.060
	Sig. (2-tailed)	.963	.291		.591	.714
	N	40	40	40	40	40
Third ventricle	Pearson Correlation	-.150	.525**	-.088	1	.295
	Sig. (2-tailed)	.354	.001	.591		.065
	N	40	40	40	40	40
Temporal horn	Pearson Correlation	-.086	.522**	.060	.295	1
	Sig. (2-tailed)	.599	.001	.714	.065	
	N	40	40	40	40	40

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Figure 32

Correlation between CSF Pressure and Evans Index

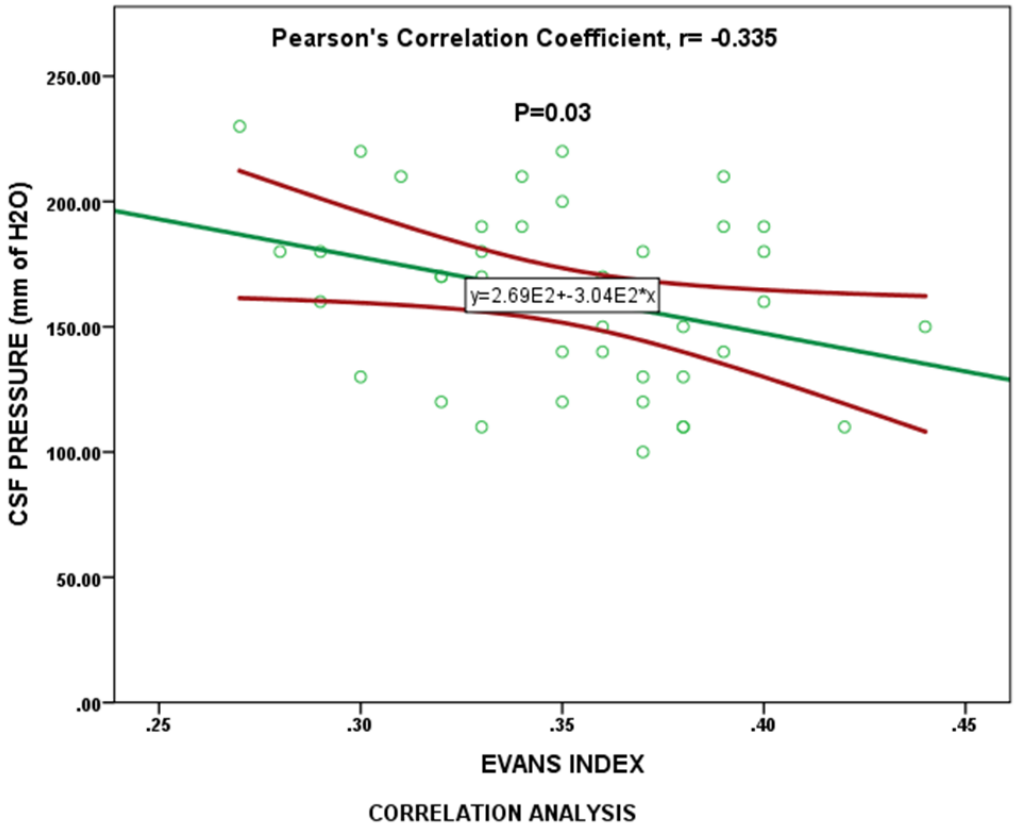

Table 27

CORRELATIONS

		EVANS INDEX	TEMPORAL HORN
EVANS INDEX	Pearson Correlation	1	.522**
	Sig. (2-tailed)		.001
	N	40	40
TEMPORAL HORN	Pearson Correlation	.522**	1
	Sig. (2-tailed)	.001	
	N	40	40

**. Correlation is significant at the 0.01 level (2-tailed).

Figure 33

CORRELATION BETWEEN SIZE OF TEMPORAL HORN AND EVANS INDEX

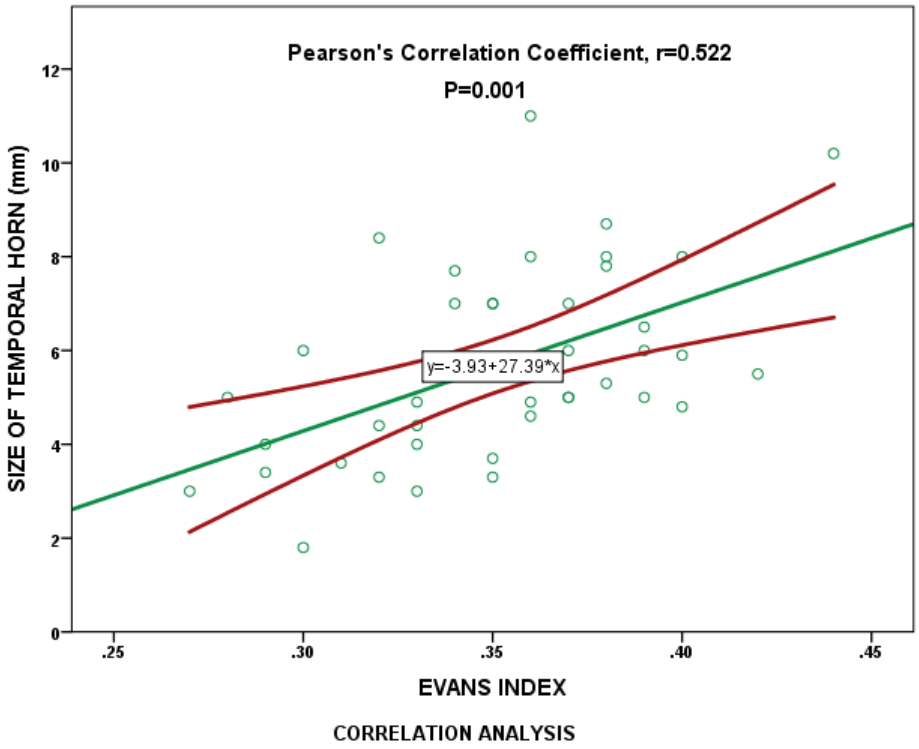

Table 28

PEARSON'S CORRELATION ANALYSIS

		CALLOSAL ANGLE	TEMPORAL HORN
CALLOSAL ANGLE	Pearson Correlation	1	.060
	Sig. (2-tailed)		.714
	N	40	40
TEMPORAL HORN	Pearson Correlation	.060	1
	Sig. (2-tailed)	.714	
	N	40	40

Figure 34

CORRELATION BETWEEN MAXIMUM DIAMETER OF TEMPORAL HORN AND CALLOSAL ANGLE

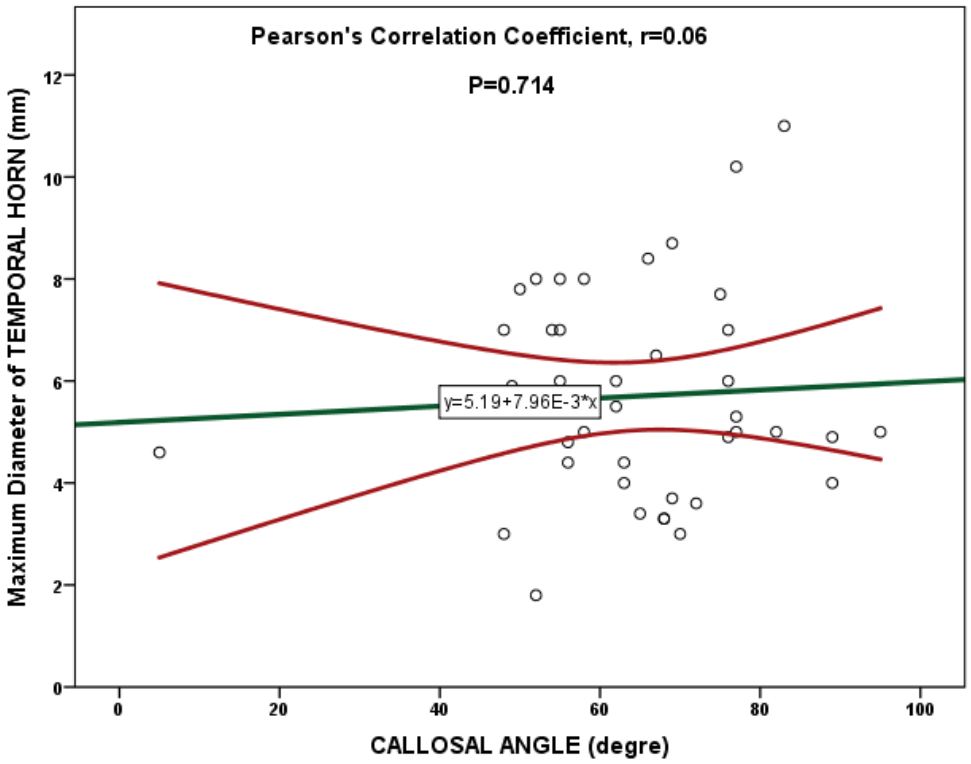

Table 29

CORRELATION BETWEEN DWMH AND PVH

		DWMH	PVH
DWMH	Pearson Correlation	1	.459**
	Sig. (2-tailed)		.003
	N	40	40
PVH	Pearson Correlation	.459**	1
	Sig. (2-tailed)	.003	
	N	40	40

**. Correlation is significant at the 0.01 level (2-tailed).

Figure 35

Correlation between DWMH and PVH

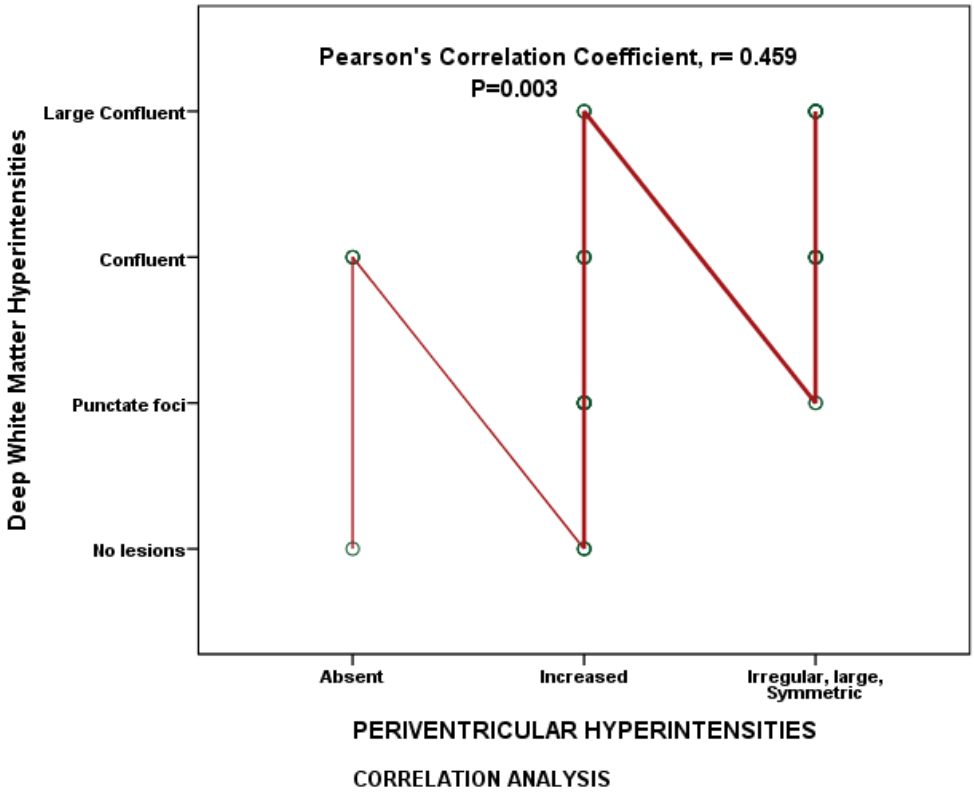

Table 30

CORRELATIONS

		FOCAL LV BULGE	**COMPLETE DESH**	**TEMPORAL HORN**
FOCAL LV BULGE	Pearson Correlation	1	.273	.073
	Sig. (2-tailed)		.092	.656
	N	40	39	40
COMPLETE DESH	Pearson Correlation	.273	1	.317*
	Sig. (2-tailed)	.092		.049
	N	39	39	39
TEMPORAL HORN	Pearson Correlation	.073	.317*	1
	Sig. (2-tailed)	.656	.049	
	N	40	39	40

*. Correlation is significant at the 0.05 level (2-tailed).

Figure 36

Correlation between complete DESH and max diameter of Temporal Horn

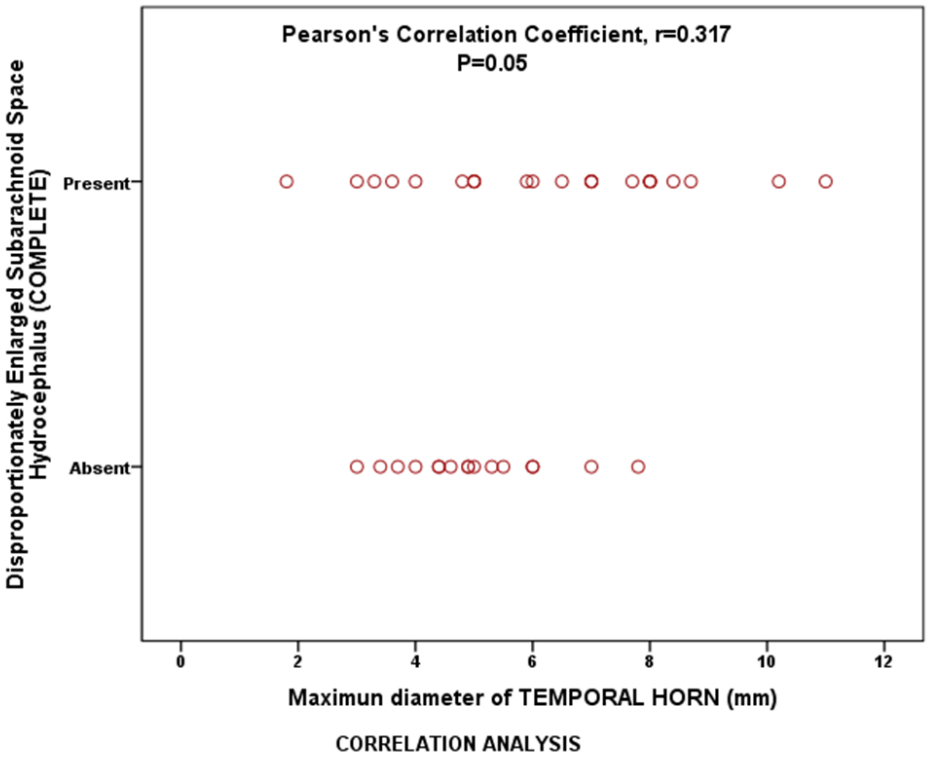

Table 31

CORRELATIONS

		CSF TAP	GAIT ATAXIA	URINARY INCONT	DEMENTIA
CSF TAP	Pearson Correlation	1	-.388*	-.282	-.258
	Sig. (2-tailed)		.013	.078	.108
	N	40	40	40	40
GAIT ATAXIA	Pearson Correlation	-.388*	1	.126	.192
	Sig. (2-tailed)	.013		.439	.234
	N	40	40	40	40
URINARY INCONT	Pearson Correlation	-.282	.126	1	-.036
	Sig. (2-tailed)	.078	.439		.824
	N	40	40	40	40
DEMENTIA	Pearson Correlation	-.258	.192	-.036	1
	Sig. (2-tailed)	.108	.234	.824	
	N	40	40	40	40

*. Correlation is significant at the 0.05 level (2-tailed).

Figure 37

CORRELATION BETWEEN GAIT ATAXIA AND CSF PRESSURE

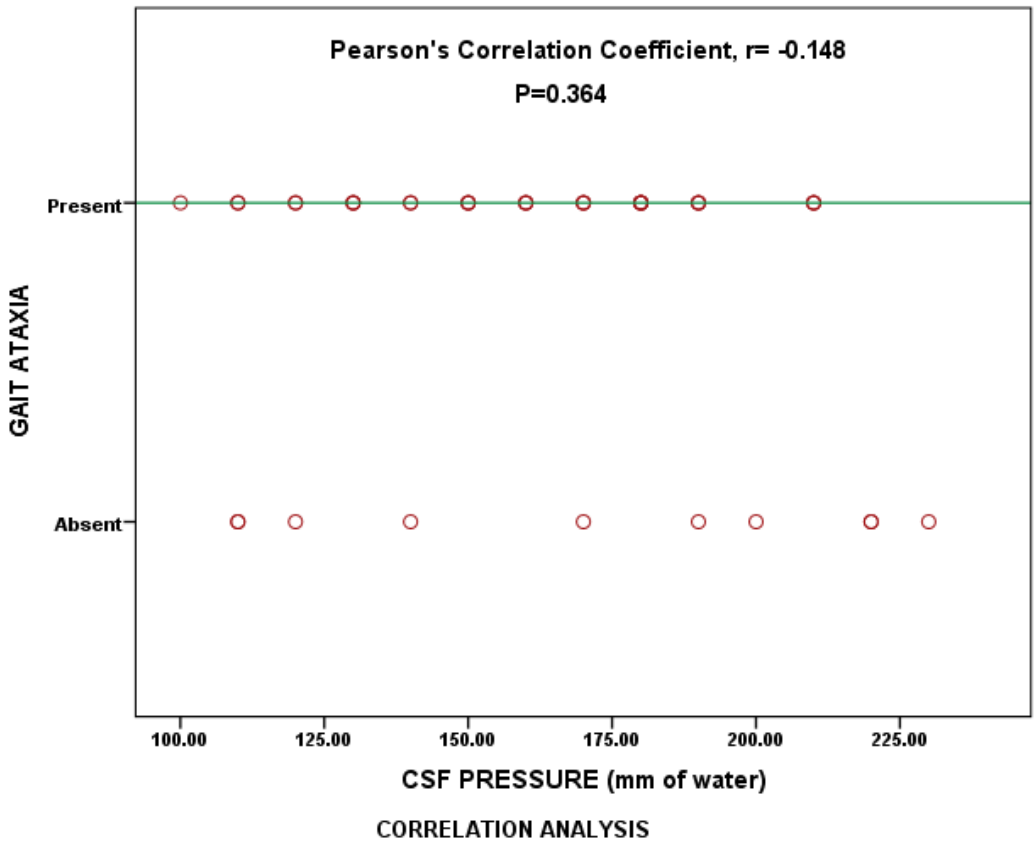

Table 32

DESCRIPTIVE STATISTICS

	N	Range	Minimum	Maximum	Mean	Std. Deviation
AGE(YEARS)	40	43.00	42.00	85.00	65.7750	9.19166
EVANS INDEX	40	.17	.27	.44	.3518	.03935
CALLOSAL ANGLE	40	90	5	95	64.68	15.516
THIRD VENTRICLE	40	6	7	13	10.40	1.583
TEMPORAL HORN	40	9	2	11	5.70	2.065
CSF PRESSURE	40	130.00	100.00	230.00	162.0000	35.67661
Valid N (listwise)	40					

DISCUSSION

DISCUSSION

Normal pressure hydrocephalus (NPH) is as a syndrome of gait ataxia, dementia, and incontinence associated with normal CSF pressures and dilated ventricles. This condition predominantly affects the elderly population. [71]

Imaging techniques are increasingly being used for diagnostic and prognostic evaluation of NPH due to lack of a single standard test. [72-73] Computed tomography (CT) and magnetic resonance imaging (MRI) shows ventricular enlargement disproportionate to cerebral atrophy, with associated ballooning of frontal horns, periventricular hyperintensities, thinning and elevation of the corpus callosum, and widening of temporal horns without evidence of hippocampal atrophy. Although CT findings alone can suggest a diagnosis of NPH, MRI may be more useful for disclosing associated pathologies (such as cerebrovascular disease), prognostic signs, avoiding exposure to ionizing radiations. [74]

The present cross section observational study evaluated imaging characteristics (CT & MRI) in patients with normal pressure hydrocephalus at a tertiary care center in the southern part of the Indian sub-continent. The outcome variables evaluated were the Evans index, Callosal angle, sulci, sylvian fissure dilation, diameters of third ventricle and temporal horns of lateral ventricle, Flow voids through aqueduct of sylvius, deep white matter hyper intensities, periventricular hyperintensity, disproportionately enlarged subarachnoid hydrocephalus (DESH) and focal bulging of lateral ventricles. The impact of independent variables like age and gender on these indices were also evaluated.

Evan's Index (the ratio which compares the maximum width of the frontal horns of the lateral ventricle to the maximum transverse diameter of the inner table of the skull) is an important parameter for diagnosis of NPH and ventriculoperitoneal shunt surgery follow-up.[75-76]

Samuel et al conducted a case control study to define the value of quantitative MRI biomarkers (Evans' Index (EI), Aqueduct Flow Rate, and Apparent Diffusion Coefficient) in idiopathic NPH. Nine patients (age, 57–79 years; mean, 70.2 years) with a clinical diagnosis of idiopathic NPH were studied and compared with nine age and gender-matched controls. The authors found a significant difference in EI between cases and controls. For patients presenting with signs and symptoms of NPH, readings on MRI greater than 0.3, 10 mL/ min, and 10.65 104 mm2 /s for EI, peak diastolic flow rate, and ADC, respectively, further reinforces the diagnosis. The mean EI of 0.35±0.04 in our study was comparable to the observations by Samuel and co-authors. [77]

Arun Kumar et al evaluated EI in South Indian population using computed tomography. One hundred subjects (5 to 90 years) with normal CT brain were analyzed retrospectively. There were 54 males and 46 females. The authors found that the mean EI was 0.27 ± 0.04 in males, 0.26 ± 0.03 in females, respectively. No significant statistical difference was observed in the EI between males and females. However, with advancing age, mild increase in Evan's index was seen. In our study, no significant difference (P=0.342) was observed in EI between males and females. [78]

Brix et al evaluated 534 subjects (53% women) between 65-84 years of age for ventricular enlargement, radiologically. Out of these, 226 patients had Alzheimers (AD) and 308 were healthy elderly controls (CTR). The cut-off for

pathological ventricular enlargement was estimated from healthy elderly categorized into age groups of 5 years range and defined as EI 97.5 percentile (mean+2SD). Cut-off values were tested on patients with Alzheimer's disease and a small sample of patients with probable idiopathic normal pressure hydrocephalus (NPH) to assess the sensitivity. The authors found that EI increases with age in both CTR and AD, and the overall EI for women were lower than for men ($p<0.001$). When applying the proposed cut-offs for EI in men and women aged 65-84, they differentiated between NPH and CTR with a sensitivity of 80% and for different age and sex categories of AD and CTR with a sensitivity and specificity of 0-27% and 91-98%, respectively. The wide range of EI measurement in elderly suggest that a cut-off value of 0.3 cannot differentiate between normal and enlarged ventricles. [79]

Callosal angle (the angle between the lateral ventricles) is considered as a prognostic indicator in NPH. In NPH, it is usually between 40º and 90º. Virhammar et al retrospectively evaluated preoperative MRI brain scans in 109 patients who had undergone shunt surgery for NPH. The patients were examined clinically before surgery and at 12 months postoperatively. Shunt responders had a significantly smaller mean preoperative CA compared with non-responders: 59° (95% CI 56°-63°) versus 68° (95% CI 61°-75°) ($p < 0.05$). A callosal angle cut-off value of 63° showed the best prognostic accuracy. Although, monitoring response to shunt surgery was not an objective of the present study, a mean Callosal angle of 64.7±15.5 degrees agreed to the study by Virhammar et al. [80]

Wide temporal horns are predictors of a positive shunt outcome in patients with NPH. Evaluation of temporal horns and hippocampus may distinguish hydrocephalic enlargement of the ventricle, as occurs in NPH from

ventriculomegaly secondary to cerebral atrophy. Kojoukhova et al retrospective evaluated brain CT or MRI scans in a cohort of 390 patients with suspected NPH. Volumes of cerebrospinal fluid compartments (lateral ventricles, sylvian and supra-sylvian subarachnoid spaces and basal cisterns) were visually assessed. Quantitative markers like EI, the modified cella media index, mean width of the temporal horns and callosal angle were measured. Mild disproportion (OR 2.6, CI 95 % 1.4-4.6, $P = 0.001$) and narrow temporal horns (OR per 1 mm 0.91, CI 95 % 0.84-0.98, $P = 0.014$) were associated with an NPH diagnosis. NPH was more likely in patients with severe volumetric disproportion between the supra-sylvian and sylvian subarachnoid spaces than in those without disproportion (OR 7.5, CI 95 % 4.0-14.1, $P < 0.0001$). [81]

Aqueduct flow void is a loss (increased hypo-intensity) of signal seen within the aqueduct and neighbouring third and fourth ventricles, particularly on T2-weighted MRI images may or may not predict outcome after shunt surgery. In NPH, there is greater outflow of CSF through the aqueduct with subsequent increase in signal loss (void sign). Krauss et al investigated the predictive value of CSF flow void on outcome after shunting in a prospective series of patients with idiopathic NPH. The degree and extension of CSF flow void was examined on T2-weighted magnetic resonance imaging scans of 37 elderly patients with idiopathic NPH who underwent subsequent shunting and similar number of age-matched controls. The authors found that aqueduct CSF flow void did not differ significantly between cases and controls. The extension of the CSF flow void, which was significantly greater in the NPH group. However, CSF flow void did not correlate significantly with surgical outcome. The authors suggested that CSF flow

void findings on conventional magnetic resonance imaging scans may not aid in making the decision to offer shunting in patients with idiopathic NPH. [82]

Hashimoto et al conducted a multicenter prospective study (Study of Idiopathic Normal Pressure Hydrocephalus on Neurological Improvement: SINPHONI) to evaluate the utility of the MRI-based diagnosis in NPH. Hundred patients (60 and 85 years) with one or more of symptoms (gait, cognitive, and urinary problems) and MRI evidence of ventriculomegaly and tight high-convexity and medial subarachnoid spaces received VP shunt using the height/weight-based valve pressure-setting scheme. The authors suggested that Tight high-convexity and medial subarachnoid spaces, and enlarged Sylvian fissures with ventriculomegaly, defined as disproportionately enlarged subarachnoid-space hydrocephalus (DESH), are worthwhile for the diagnosis of NPH. [83]

Periventricular signal changes may be associated with subcortical vascular encephalopathy (also with lacunar infarctions) in NPH. Periventricular and deep white matter hyperintensities were observed in 90% NPH patients in our study. Their diagnostic and predictive value in normal pressure hydrocephalus (NPH) is unclear. Tullberg et al conducted MR imaging in 34 patients with NPH before and after shunt surgery. T2-weighted turbo spin-echo sequences and coronal T1-weighted sequences were performed on a 0.5-T imager. The other variables assessed were cortical and subcortical lacunar infarctions, the flow void sign, and the width of the third and lateral ventricles were registered. Gait ability need for sleep, urinary incontinence, living conditions, and psychometric test performance were also assessed pre- and postoperatively. The authors suggested that presence of DWMH or subcortical lacunar infarctions in NPH did not predict a poor outcome from shunt

surgery and should not be used as exclusion criteria for shunting. No MR imaging findings could predict outcome of shunt surgery in patients with NPH. Clinical improvement after surgery was associated with reduction in the irregular type of PVH located around the frontal horns. [84]

The CSF tap test (CSF-TT) is a diagnostic tool used to select patients with idiopathic NPH for shunt surgery. Quantitative testing of gait and cognitive functions are done before and after the drainage of 40-50 ml lumbar CSF. In the present study, 62.5% patients had a positive CSF tap test. In a study by Virhammar et al, forty patients (21 men and 19 women) under evaluation for NPH underwent a tap test. Standardized gait analyses were performed before and 2, 4, 6, 8 and 24 h after the TT and repeated twice on every occasion. The test was responsive in 67.5% patients. These results agreed with our study. [85]

Correlations often exist between radiological markers in idiopathic NPH. Decrease in ventricle size is usually not detectable postoperatively either by visual assessment or by measuring the Evans index in NPH patients. Virhammar et al investigated the correlation between the callosal angle and ventricular volume after shunt surgery. Magnetic resonance imaging of the brain was performed before and 3 months after shunt surgery in 18 patients with NPH. The CA and Evans index were measured on T1-weighted 3D MR images, and ventricular volume contralateral to the shunt valve was measured with quantitative MRI. Postoperative change of CA showed a negative but significant correlation with change of ventricular volume ($r = -0.76$, $p < 0.01$). The authors suggested that non-invasive radiological markers like CA should be evaluated further as an indirect method to determine shunt function in patients with NPH. [86]

Several correlations between imaging findings were observed in our study. Significant correlations were seen between markers of ventricular width and Evans index, temporal horn, and size of third ventricle, callosal angle and focal bulge of left ventricle. DESH correlated with callosal angle and focal LV bulge. Similar correlations were observed by Virhammar et al in a retrospective analysis of 108 patients with idiopathic NPH. The authors suggested that a small callosal angle, wide temporal horns, and occurrence of disproportionately enlarged sub-arachnoid space hydrocephalus are common in patients with idiopathic normal pressure hydrocephalus and were significant predictors of a positive shunt outcome. [87]

SUMMARY

Conclusion

CONCLUSION

A cross sectional observational study was done in the department of Radiodiagnosis, Sree Mookambika Institute of medical Sciences, Kulasekharam, Tamil Nadu India, from July 2016 to April 2018. Patients with features of normal pressure hydrocephalus according to consensus criteria who underwent CT and MRI evaluation of brain using T1W, T2W, FLAIR, ADC, DWI, GRE, D CISS sequences were assessed. The study has come up with the following observations.

Computed tomography (CT) and Magnetic resonance imaging (MRI) show ventricular enlargement disproportionate to cerebral atrophy, with associated ballooning of frontal horns, periventricular hyperintensities, thinning and elevation of the corpus callosum, and widening of temporal horns without evidence of hippocampal atrophy in NPH. Although diagnosis can be made based on CT findings alone, MRI is more accurate for disclosing associated pathologies such as grading of PVH and DWMH , transport sulci , and presence and location of flow voids , and presence of aqueductal stenosis and for detecting NPH typical signs of prognostic value, besides avoiding exposure to ionizing radiation.

- The mean Evan's index of 0.35±0.04 agreed with the standard diagnostic criteria and that with other studies.
- Evan's index did not differ significantly between males and females.
- A mean Callosal angle of 64.7±15.5 degrees agreed to other studies.
- A significant correlation was observed between ventricular width and Evans index, temporal horn, and size of third ventricle, callosal angle and focal

bulge of left ventricle. DESH correlated with callosal angle and focal LV bulge.

- DWMH and PVH are found to be more in our population of patients with NPH as compared to other studies, which needs further investigation.
- Complete DESH was found in 59 % , Incomplete DESH was found in 27.5% and Non DESH in 15% of patients in my study.

LIMITATIONS

LIMITATIONS OF STUDY

The shortcomings of the present study were a small sample size of patients. Second, NPH patients were not evaluated for shunt surgery. Consequently, MRI markers could not be evaluated for suitability for shunt surgery. Thirdly the advanced MRI techniques like CSF flow studies and diffusion tensor imaging was not performed.

SUMMARY

Normal pressure hydrocephalus remains a controversial entity with often ambiguous imaging findings. It is classically characterised by the triad of gait apraxia, urinary incontinence, and dementia, although not all patients with the condition have all three. On imaging, it can be characterised both on CT and MRI by enlarged lateral and third ventricles out of proportion to the cortical sulcal enlargement. There is no standard gold test to diagnose and monitor response to shunt surgery in normal pressure hydrocephalus. Often, clinical tests like CSF-Tap tests are used in conjunction with radiology for diagnostic and prognostic evaluation. MRI is the best modality to image anatomical changes and can further support the diagnosis with CSF flow studies.

In conclusion, imaging characteristics are very much similar to already described with western population.

The various pattern studied in NPH correlates well with each other.

DWMH and PVH are found to be more in our population of patients with NPH as compared to other studies, which needs further investigation.

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APPENDIX

APPENDIX – 1

SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES

KULASEKHARAM

RESEARCH COMMITTEE

CERTIFICATE

This is to certify that The Research Protocol Submitted by DR SPARSH YADAV

Faculty / Post Graduate from Department of RADIODIAGNOSIS

Titled IMAGING CHARACTERISTICS IN PATIENTS WITH NORMAL PRESSURE HYDROCEPHALUS AT TERTIARY HEALTH CARE HOSPITAL

is approved by the Research Committee.

Chair Person

Prof. & H.O.D.
Dept. of Bio-Chemistry
Sree Mookambika Institute of Medical Sciences
Kulasekharam 629 161

Convenor

Prof. & H.O.D.
Dept. of Physiology
Sree Mookambika Institute of Medical Sciences
Kulasekharam 629 161

Date :

APPENDIX – 2

INSTITUTIONAL HUMAN ETHICS COMMITTEE

SREE MOOKAMBIKA INSTITUTE OF MEDICAL SCIENCES,
KULASEKHARAM, TAMILNADU

Communication of Decision of the Institutional Human Ethics Committee(IHEC)

SMIMS/IHEC No: 1 /Protocol no: 45 / 2016

Protocol title: IMAGING CHARACTERISTICS IN PATIENTS WITH NORMAL PRESSURE HYDROCEPHALUS AT TERTIARY HEALTH CARE HOSPITAL
Principal Investigator: Dr. Sparsh Yadav
Name& Address of Institution: Department of Radiodiagnosis Sree Mookambika Institute of Medical Sciences, Kulasekharam ☑ New review ☐ Revised review ☐ Expedited review
Date of review (D/M/Y): 15.12.2016 Date of previous review , if revised application:
Decision of the IHEC: ☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestions/ Reasons/ Remarks:
Recommended for a period of :

Please note*

- Inform IHEC immediately in case of any Adverse events and Serious adverse events.
- Inform IHEC in case of any change of study procedure, site and investigator
- This permission is only for period mentioned above. Annual report to be submitted to IHEC.
- Members of IHEC have right to monitor the trial with prior intimation.

Reneegalyangadhar

Signature of Member Secretary IHEC

APPENDIX- 3

CASE RECORD FORM

1. Serial no :

2. Date :

3. Name (Optional) :

.....

4. Age in years :

5. Address & Phone no :

.....

.....

6) Sex : Male ☐ Female ☐

7) Presenting symptoms :

8) Past history :

9) Clinical diagnosis:

11) Dementia:

12) Gait abnormality:

13) Urinary incontinence/Urgency:

14) Cognitive impairment/ deficit:

15) Evans index

16) Callosal angle

17) Dilatation of Sylvian fissure:

18) Diameters of third ventricle and temporal horns of lateral ventricle:

19) Narrow sulci:

20) Flow voids through aqueduct of sylvius:

21) Deep white matter hyper intensities:

22) Periventricular hyper intensity:

23) Disproportionately enlarged Subarachnoid hydrocephalus (DESH),

24) Focal bulging of lateral ventricles.

APPENDIX- 4

MASTER CHART

S.NO	OP/IP NUMBER	AGE	SEX	EVANS INDEX	CA	NARROW SULCI (0-2)	SF DILATATION (0-2)	COMPLETE DESH (0-1)	INCOMPLETE DESH (0-1)	NON DESH (0-1)	FOCAL BULGE OF LV (0-1)	THIRD VENTRICLE (mm)	TEMPORAL HORN (mm)
1	18023917	71	2	0.36	83°	2	2	1	0	0	1	10.1	11mm
2	18023713	70	1	0.35	68°	2	1	1	0	0	1	10.2	3.3mm
3	17087025	60	1	0.39	58°	1	1	1	0	0	0	10.7	5mm
4	1712594	69	1	0.29	65°	0	0	0	0	1	1	9	3.4mm
5	17196040	75	2	0.35	76°	2	2	1	0	0	1	9.6	7mm
6	1711516	70	1	0.4	58°	2	2	1	0	0	1	12	8mm
7	1711598	58	2	0.32	63°	0	1	0	1	0	0	11	4.4mm
8	17064869	55	2	0.36	52°	0	1	0	1	0	0	9	4.6mm
9	1726965	80	2	0.3	114°	1	2	1	0	0	0	7	1.8mm
10	162142165	55	2	0.33	70°	1	2	1	0	0	1	11	3mm
11	1708819	63	1	0.39	62°	1	0	0	1	0	0	12	6mm
12	1729216	42	1	0.36	52°	2	2	1	0	0	0	11	8mm
13	17083534	52	1	0.35	55°	0	2	0	1	0	1	10	7mm
14	17095393	70	1	0.42	62°	2	0	0	1	0	0	11	5.5mm
15	17055280	52	2	0.37	48°	2	2	1	0	0	0	13	7mm
16	17086670	55	1	0.3	76°	0	1	0	1	0	0	10	6mm
17	14019890	60	2	0.28	95°	0	0	0	0	1	1	8	5mm
18	1721152	80	1	0.44	77°	2	2	1	0	0	1	13	10.2mm
19	17077481	64	2	0.38	55°	1	2	1	0	0	0	11	8mm

20	17039038	60	2	0.39	67°	2	2	1	0	0	1	12	6.5mm
21	1711763	75	1	0.37	77°	1	1	1	0	0	1	11	5mm
22	17067532	85	1	0.33	56°	2	0	0	1	0	1	13	4.4mm
23	17838566	69	2	0.34	54°	1	1	1	0	0	1	11	7mm
24	17077481	53	1	0.27	48°	0	0	0	0	1	0	8	3mm
25	17009929	78	2	0.36	89°	0	0	0	0	1	0	12	4.9mm
26	1721474	70	1	0.37	55°	2	2	1	0	0	1	13	6mm
27	17123681	58	2	0.32	68°	0	1		1	0	1	10	3.3mm
28	17172116	60	1	0.33	63°	1	1	1	0	0	0	13	4.2mm
29	15047448	72	2	0.37	82°	1	1	1	0	0	1	9	5mm
30	1632880	80	1	0.38	77°	2	0	0	1	0	0	10	5.3mm
31	1710610	70	2	0.32	66°	2	2	1	0	0	1	9	8.4mm
32	15047448	72	2	0.31	72°	1	1	1	0	0	1	11	3.6mm
33	17121328	65	1	0.38	50°	0	2	0	1	0	0	9.6	7.8mm
34	17069924	66	1	0.33	76°	0	0	0	0	1	0	8.7	4.9mm
35	1705171	68	2	0.4	56°	2	2	1	0	0	0	9.9	4.8mm
36	1704696	65	1	0.38	69°	1	1	1	0	0	0	11	8.7mm
37	17083220	64	1	0.29	89°	0	0	0	0	1	1	9	4mm
38	13110751	72	2	0.35	69°	0	2	0	1	0	1	8	3.7mm
39	17069924	66	1	0.4	49°	2	2	1	0	0	1	11.3	5.9mm
40	1701494	62	2	0.34	75°	1	1	1	0	0	1	8	7.7mm

DWMH (0-3)	PVH (0-2)	TRANSPORT SULCI (0-1)	FLOW VOIDS (0-3)	AQUEDUCTAL STENOSIS(0-1)	CSF PRESSURE (mm h20)	CSF TAP TEST	URINARY CONTINENCE(0-1)	DEMENTIA (0-1)	GAIT ATAXIA (0-1)	VP SHUNT
1	1	0	1	0	160	1	1	1	1	NP
0	1	0	1	0	140	2	0	1	1	NP
2	2	0	0	0	210	2	0	1	1	NP
2	2	1	2	0	180	2	1	1	1	NP
0	0	0	3	0	200	1	1	1	0	NP
1	1	1	1	0	180	1	0	1	1	NP
1	1	0	2	0	120	1	1	1	1	NP
2	2	0	3	0	170	2	1	1	0	NP
1	1	1	1	0	130	1	1	1	1	NP
3	2	0	1	0	180	1	1	1	1	NP
2	1	0	0	0	190	2	0	1	1	NP
0	1	1	0	0	140	1	1	1	1	PERFOMED
1	2	0	0	0	220	1	1	1	0	NP
1	2	0	3	0	110	1	1	1	1	NP
3	2	0	0	0	100	1	0	1	1	NP
1	1	0	0	0	220	2	1	1	0	NP
2	2	0	0	0	180	1	1	1	1	NP
2	1	0	2	0	150	1	1	1	1	NP
0	1	0	3	0	130	1	1	0	1	NP
1	1	0	2	0	140	2	1	0	0	NP
3	1	1	2	0	120	1	1	1	1	NP
3	1	1	2	0	110	2	0	1	0	NP
2	1	0	3	0	210	1	0	1	1	NP

3	2	0	0	0	230	2	0	0	0	NP
2	1	0	2	0	150	1	1	1	1	NP
3	1	1	2	0	180	1	1	1	1	PERFOMED
2	0	0	0	0	170	1	0	1	1	NP
1	1	0	0	0	190	2	0	1	0	NP
3	2	0	0	0	130	1	0	1	1	NP
1	1	0	1	0	150	2	1	1	1	NP
3	2	0	1	0	170	2	1	1	1	NP
3	2	0	0	0	210	1	1	1	1	NP
2	0	1	0	0	110	1	1	1	0	NP
2	0	0	2	0	170	2	1	1	1	NP
3	2	0	3	0	160	1	1	1	1	PERFOMED
3	2	0	0	0	110	1	1	1	1	NP
3	2	1	0	0	160	1	1	1	1	NP
3	2	0	1	0	120	2	0	1	0	NP
2	2	0	0	0	190	2	1	0	1	NP
2	2	0	1	0	190	1	1	1	1	NP